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THE

MEDICAL JOURNAL OF AUSTRALIA

Vol. I.—10TH YEAR.

SYDNEY: SATURDAY, MAY 19, 1923.

No. 20.

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NOTES ON PSYCHO-THERAPEUTIC PRACTICE¹

By J. V. McAREE, M.B., B.S. (ADELAIDE),
Adelaide.

SOME years ago I gave a paper on suggestive therapeutics. I maintained that suggestion was the most useful of the various methods of psychiatry, whether it is used with or without hypnosis. Personally I prefer the latter, as it usually aids suggestion in becoming fixed, so that it either displaces a harmful acquired idea or rouses the curative mechanism of the brain which has been inactive or sluggish, often from a fixed auto-suggestion of impotency.

One of the great difficulties which both medical men and the laity had in accepting the reality of suggestion was that it seemed impossible to get a scientific basis for its action. That a chemical swallowed or knife wielded would be beneficial seemed much more rational, but how spoken words could do any good was beyond comprehension. Yet, every day one sees the effect of the psychic on the psychic, just as we see the physical acting on the physical.

We see the effect of bad news on a person; how it can not only depress, but actually cause both

physical and mental breakdown. There is no doubt of a powerful force acting here, which, though invisible and intangible, is far-reaching in its effects. The doctrine of suggestion is but an elaboration of this. All gregarious animals are markedly influenced by their fellows. A rabbit stamps its foot at the sight of danger and at the suggestion the whole warren stampedes for safety. In man the animal of subconscious mind is open to receive suggestion which will either stimulate or modify its views. I consider the basis of all modern treatment is suggestion, hypnosis being merely an increase of the natural suggestibility. Theoretically everybody is hypnotizable, except the insane, but owing to various factors many people, chiefly from auto-suggestion, do not reach the stage commonly called hypnosis.

This led to a school called the persuasionists, who wrongly decried hypnotic treatment, saying that persuasion was the only thing that counted. The thousands of cures reported by such reliable authorities as Bernheim and Liébault should have been sufficient to have proved that hypnotic suggestion properly used was most beneficial.

The fact of the matter is, as I have said before, that there are some people who, while prevented by auto-suggestion from being hypnotized, will accept suggestion, especially when in a suitable relaxed state. As an example of this I

¹ Read at a meeting of the South Australian Branch of the British Medical Association on March 29, 1923.

may instance a woman who had vomited and had been confined to her bed for two days each time she was unwell for over a period of twelve years. I tried hypnosis, but she was a bad subject, opening her eyes frequently to show she was not asleep. So I told her to keep her eyes closed and listen to what I said. I have her a suggestion that she would get better and so forth. At the close of the sitting she opened her eyes and assured me that she had not been off. I myself could not see the slightest evidence either. She had six similar sittings. That was over two years ago and she has never vomited or had the slightest trouble since.

Unfortunately psychiatry has not only been attacked from without but also from within. The different schools have exceeded healthy criticism of each other and in many cases proclaimed their views as the only one that is true. Examples may be cited in Boris Sidis and Professor Forel.

The point to consider is that all have had undoubted successes and as such deserve our consideration. It will be the object of my paper here to try to reconcile the apparently divergent methods.

As regards psycho-analysis I use it very often in conjunction with treatment by hypnosis, especially when there is a definitely sexual element to start with. There is no doubt that the greater portion of our dreams are sexual, though the word has to be used in a far broader sense than is usually implied. I could never agree in believing that all the dreams were so, because theoretically it does not seem logical to me and because it is so easy unconsciously to suggest what is wanted. Thus I would imagine that there would be few of Freud's patients who did not have a smattering of the sexual theory before they came to him and consequently it might be possible that an influence association might be formed.

When we get down to bedrock as regards the instincts of the subconscious or animal mind, we may take it that the first is to live and the second to be happy.

The preservative instinct branches into two other instincts. One is to continue life after death, which is the basis of all religion, and the second is to continue our life through our offspring, which is the sexual instinct. These instincts claim constant satisfaction. As regards the sexual one, owing to gregarious reasons its outlet is constantly being inhibited, especially amongst the Caucasian races, who regard sex as something shameful. Now in sleep the censor (as the controlling part of the brain is called) relaxes and the pent-up sexual desires are allowed to escape, either indirectly by means of symbolization or directly as a sexual dream. When we look on sleep mainly as a means of rest and recuperation, it is only to be expected that the psychic should be refreshed as well as the physical.

Freud, as I have said, considered everything as sexual and, looking for it, he undoubtedly found it in some cases by suggestion. When the war came, we had a chance of seeing the same thing happen to the self-preservative instinct when it was out-

raged by discipline and courage. In the light of this we can understand how many cases of insanity or mental breakdown are brought about by this. If a man is in good health and subjected to the ordinary strains, nothing much happens, but if he has some defect owing to heredity in the development of his censor or his instincts, the strain is often felt and is shown as a hysterical person in whom the emotional force is converted into a motor or sensory disability, when the dreams are insufficient to absorb all.

Especially after such diseases as influenza do we see the loss of control and the resultant melancholia. Melancholia is especially prone to come on in women, especially near the change of life, and in some patients I have attended, I feel convinced that the mental attitude, both subconscious and conscious, with which life is regarded as this time is a big factor. I will now quote a case in which I used both suggestion and psycho-analysis:

Some years ago, about a month after confinement, Mrs. W. became insane. She several times attempted suicide by swallowing a bottle of "Phenacetin" tablets and on another occasion by jumping down a well. She lost all interest in her personal appearance and used to spend most of her day tearing up things. Her baby had to be sent away lest she should injure it. She was like this for nine months, after which she was sent to Adelaide, where she was put into a hospital for several months, during which Weir-Mitchell and other treatments were tried, but she became worse. It was decided to send her to the asylum, when she came to me, having been advised, I believe, by Dr. Riddle, of Kapunda.

On her arrival she was composed enough to speak to me and told me that I would never put her to sleep and, if I did, she did not believe it would do her the slightest good. However, I got her to sit in a chair and in a few minutes had her in deep somnambulism. I gave her some suggestions and on awakening she said she felt better. A couple of nights after that I put her into a deep sleep. Whilst she was sleeping peacefully, the window rattled slightly. The result was dramatic, as she shrieked out and tried to start up. I could see she had some hidden complex which the rattling of the window had aroused. So I proceeded with psycho-analysis. I gave her the word rattle and then wind. She immediately mentioned a certain country town; then operation. To make a long story short, she told me that a month after her confinement she had to undergo a curettage. This was done without any anæsthetic. She was a peculiarly sensitive woman and the shame attending the feeling of exposure before a number of people acting on her already weakened brain started a melancholy feeling which she could not shake off. This formed the nucleus around which her melancholia was built.

She noticed at the time that the wind was rattling the window of the operating room. Her husband, who was present, then stated that he had noticed during the whole time of her illness that her melancholia was always aggravated on stormy days. I then gave her a special suggestion that as she was being cured on a stormy night, she would in the future look on the wind and storm in an opposite way. I then gave her the word "baby" and her associated word was "unhappy." This she said was because she really wanted to be near it, but had been prevented from loving it by some power over which she had no control.

I told her that all would be well and that she would go home and nurse it. I then awakened her. Her husband, who was standing beside her, said: "What did the doctor say?" She replied: "He had not yet spoken, as I have only just sat down." She had been in the chair, as a matter of fact, for half an hour.

Nothing further was said, but a few days afterwards she was with her husband in a room when a storm suddenly came with thunder and lightning. She immediately went to the window, lifted up the blind and remained

gazing out. The husband asked her why she did so. She replied she did not know why, except that it gave her comfort. Previously she had always cowered on the floor when the storm was on. He then asked her about her baby and she said she wanted to go home and nurse it, which she did. She has remained perfectly well ever since. In fact, I have attended her for two other confinements since.

The above case would come under the sexual group. I will now quote a case illustrating affection of the self-preservative instinct:

Mr. O., of Renmark, suffered from shell-shock in France, having been buried with his machine-gun by a shell explosion. On regaining consciousness he found that he had no control over his nerves. He trembled and had insomnia, nightmares and subconscious day-terrors, the latter coming on quite suddenly, without apparent cause. He was invalided back to South Australia and was treated for three years at Keswick without benefit. He was finally sent to me. I put him into full somnambulism in a few minutes. I then questioned him as regards his dreams and he told me that he dreamed constantly that he hurled mud at the Germans. The association for mud was "unarmed." Finally I got out of him that for some days before he was buried, he was, owing to some mistake in orders, too far advanced with his party, so that he was inadequately supported by reserves and ammunition. This, combined with the horrors he saw, was a tremendous strain on his self-preservative instinct. Above all things, he felt impotent; hence the ineffective weapons were symbolized as mud. When the explosion knocked his censor out of action, the self-preservative impulses were subconsciously diverted to form his neurosis, which had the effect of removing him from an otherwise intolerable situation. After suggesting sleep to him, control of his nerves and so forth, I awakened him. For three years previously he had never slept for more than three or four hours a night. During the night of his sitting he slept for ten hours a dreamless sleep. Every night after that he slept long and deeply. He put on a stone in weight in one month. He has now been working for two years and is quite well and strong.

It is difficult to be exact in regard to the class of patient who is likely to be benefited by suggestive treatment, especially those who are said to be insane. I used to consider that insanity was entirely resistant to such treatment, but that view, in light of some of my cases, requires revision. Personally I regard mental disease in the same way as disease with mainly physical symptoms. Physical symptoms are divided into organic and functional, whilst the disease very often has both and towards its finish the organic merge more or less insensibly into the functional. This has often been demonstrated following injuries received during the late war. From my observations I consider that purely mental symptoms, such as melancholia, mania and the like, may do the same. Consequently, in acute conditions, especially with delusions, success is not likely to be obtained, but in cases of a more functional nature which we call psychosis, the success is often dramatic. In many affections which start as real melancholia, the symptoms persist after the organic cause has been removed. These patients have done very well and I have had a number of successes. Many are not amenable, but this does not take away from the fact that others are definitely cured. Even in the more acute conditions which have not been cured, I have noticed decided improvements at times. The following illustrates what I mean:

Mr. X. was the father of a medical man. He was brought to me by his son. He was suffering from melan-

cholia of three years' duration. In addition to being depressed he suffered from anxiety which was so unreasonable that he was afraid that something terrible was going to happen. Thus, if his wife went out of the room for a few minutes, he thought that she would desert him or if she had to travel by train, he feared she might be killed. He also suffered from insomnia and constipation. He was completely incapacitated for work. I treated him at my rooms for some time with only slight improvement. I then visited him and would put him to sleep for three hours at a time, during which time I gave him curative suggestions. He showed marked improvement and got well, so that he was able to return to his business.

Leaving out the question of the more serious mental diseases, I was amazed to learn how many minor psychoses exist in the population. I am not basing my calculations on the fact that naturally I see many patients of this class who have come to me specially, because that would give a wrong impression, but upon information given me by patients who have come for some other complaint and who mentioned their particular "skeleton in the cupboard" when the subject is led up to such matters by a little judicious talking or questioning. Many patients told me they had not mentioned them to other doctors, because it seemed as if they would not be understood or that the doctor would not want to be "bothered with such eccentricities."

It would be impossible in a paper like this to refer to more than a small proportion of cases in this class. Some of the conditions are not looked upon by the patient as a psychic abnormality till the affection is diagnosed by psycho-analysis. For instance, a good looking, healthy, single woman, aged thirty, happened to mention to me that she never had the slightest conscious wish to marry. In fact, she said she had an aversion to men. As this seemed to me most unnatural, I suggested looking for the cause by psycho-analysis. I cannot go into the details here, but just mention some of her chief associations. "Marriage" gave the word "disgusting"; "water" gave the word "brook"; then came the word "hill" and "trees"; then an indescribable sense of fear. Finally she remembered that she was going home from school when she was about seven years old, she came to a lonely part through which a brook ran and around which trees were growing. A man came who committed a sexual offence which horrified and disgusted her. This she admitted had tinged all her ideas of men afterwards, though she had long ago forgotten the incident, just as in the same manner an article of food which has sickened a child, will often continue to remain nauseous for that child for the rest of its life.

People are often worried by various imperative ideas which dominate them in all possible ways. They may be compelled to entertain certain thoughts which they believe to be blasphemous or they may be compelled to perform certain actions as though they were hypnotized. Among persons affected in this way the dipsomaniacs and morphia-maniacs must be classed.

Chronic alcoholics are in a different class, as they have a constant, conscious craving for the drug. To put it in a nutshell, the former condition is a disease of the subconscious mind and the latter one of the conscious. As it has been said, the dipso-

maniac drinks because he is insane and a chronic alcoholic is insane because he drinks. I will now quote some instances to show what is possible in such cases:

Mrs. X. came to me, asking me if I could save her. She told me that she was a morpho-maniac and that she had started taking "Chlorodyne" for some ailment some years previously. She had steadily increased the amount to two bottles of "Chlorodyne" a day. On several occasions she had tried for a few hours to be without it, but found it impossible to sleep or even to be rational. She used the greater part of the household money to buy the drug. I decided to hypnotize her a few times in order to gain control before attempting to cut off the "Chlorodyne." After the third time I got full somnambulism, so I sent her over to the North Adelaide Private Hospital and ordered the withdrawal of all "Chlorodyne." That night I went over to see her and put her to sleep. She slept for thirteen hours on the first night. Next day she complained of abdominal cramps and diarrhoea. I put her to sleep again and suggested removal of the cramps, which was successful, whilst the diarrhoea gradually abated. Each night for a week I put her to sleep and, though at times she became restless, with some craving, this all disappeared after the end of the first week. Strong counter-suggestions were given her to hate "Chlorodyne." She was discharged from the hospital at the end of fourteen days. A month later she had put on over a stone in weight and twelve months afterwards she was in good health and had not touched "Chlorodyne" or any other narcotic.

In cases of this nature and of chronic alcoholism the craving is entirely conscious all the time. In dipsomania the craving is largely in the unconscious, having become so like many other habits after being practised by the conscious for many years. To illustrate I will quote the following selected from a number of others:

Twenty years ago Mr. C. was a heavy drinker, especially when he came down from a station every few months to Adelaide. This went on for ten years. He then came to Adelaide to live, resolving at the same time to give up alcohol entirely. All went well for three or four months, when suddenly he found himself drinking heavily. The curious part of it was that he had complete loss of memory for the first day, so that he did not know how he had started. The attack lasted for about a week and he was another week or two getting over the effects. He then thought he was all right, but the same thing was repeated again at short intervals for the next ten years. All these attacks were characterized by complete loss of memory of how he had started drinking. I was interested particularly in the amnesia, as it has been my experience that nearly all cases where purposeful acts have been performed with subsequent loss of memory, such as occur in natural somnambulism, insanity, dreams, forgotten acts of childhood and the like, the memory can be restored during deep hypnosis. This patient proved no exception to the rule, because, after I had got him off into somnambulism, I questioned him and took down his reply on paper. He had just recovered from a bout and for the first time he was able to tell what had happened on the first day. It appears that he was walking down Grenfell Street when a friend came and asked him to have a drink. He had a strong conscious inclination to take it, to which he yielded. As soon as the liquid had touched his tongue something seemed to snap within him. The unconscious was brought into play, a raging thirst followed which nothing could satiate. He used up what money he had in his pocket and then ran all the way to his office to get some more. He could not even wait to unlock the cash box, but broke it open and, seizing the money, rushed back to the hotel. All day he wandered from one hotel to another. He described in greatest detail the various people he met and the conversations he had, till worn out he slept in the parklands that night.

Next day he went home and from this time he dimly remembered what happened without the aid of hypnosis.

It was a continuous thirst and lifting of glass after glass to his lips.

I proceeded to give him suggestions to dislike alcohol. I also added that if he ever tried to drink alcohol, he would commence to vomit. After treating him for some time, I did not see him till a year afterwards, when he walked one evening into my surgery and said: "You were right, doctor! The craving left me entirely, but about nine months after you treated me, I had some financial worries and, being despondent, I said to myself: 'Confound the doctor, I will have a drink to cheer me up.' I tried to drink some beer, but no sooner did I bring it near my mouth than I started to vomit most violently. I tried for three days to swallow some, but the vomiting was continuous. Finally I gave it up and all inclination to take it left me. I am now delighted that I was prevented from taking it. Even now, if I pass by a public house, the smell of the beer makes me retch, so I have to hurry past or even go to the other side of the road." He is still a teetotaler after several years.

In selecting patients whom I consider suitable for treatment, I think that the first essential is that the patient must of his own wish sincerely want to be cured and not be merely forced to come to the doctor by the pressure of friends or relatives. I usually question the patient closely about this, making my decision as much from the manner in which he replies as from the actual words he uses. Dipsomaniacs are often anxious to be cured and are then prepared to undergo any inconvenience in the intervals to be properly treated.

It is always interesting to note the particular exciting causes which will precipitate a dipsomaniac attack. These may be either physical or psychic. Thus one man who had been suffering from dipsomania for years, admitted that the smell of alcohol on his brother's breath was a particular stimulant to an attack. He was in business and rightly or wrongly felt that his brother wanted him out of the way. The brother drank fairly heavily, but was never a dipsomaniac. My patient used to go "right out" for weeks at a time, being quite helpless, lying on the floor and passing motions under him. In the intervals, when he smelt the alcohol on his brother's breath, a revulsion of feeling came that the brother was able to take it without having his position in the firm endangered, whilst he might have to lose his partnership for the same reason. Curiously enough, this suggested recklessness and often started an attack or, to be more exact, a restless state, due to subconscious mentation, which lasted a few days before the attack came on. For convenience of description I may describe the alcoholic complexes in the subconscious as "powder magazines" and such exciting causes as "matches." In my suggestions I try as far as possible to find the matches and to give counter-suggestions. Thus, in this case I suggested that, whenever he smelt his brother's breath, he would feel he was showing his superiority by having his own free and as such could conquer his brother's designs. I may mention that the treatment was completely successful and the patient has now remained a total abstainer for several years, not having had the slightest inclination to take alcohol.

I will now pass on to another class of case, namely, what I call psychic or imperative diarrhoea, because of its close resemblance to purely imperative ideas:

Mr. B., aged sixty-five years, had suffered from diarrhoea for forty years, during which time he had taken hundreds of bottles of medicine without benefit. The drugs had the peculiar effect that many seemed to give temporary relief, but invariably the diarrhoea returned in a few days, rendering life a burden.

I decided to subject him to a course of sittings. This he did, with the result that the diarrhoea stopped completely. He has had on an average one solid motion daily for the last two and a half years. My theory is that he got an ordinary diarrhoea years ago and possibly it caused him discomfort and embarrassment. Being of a neurotic temperament, the fear of a repetition acted like an imperative idea does on the so-called voluntary actions and so precipitated the very thing he earnestly wished to avoid.

A somewhat similar case was one of membranous colitis with very severe pain and the passing of blood. The case had been of ten years' duration. Under hypnotic suggestion there was great improvement. The pain disappeared and the stools became normal.

I have also had cases of obstinate constipation cured by suggestion. The action is understandable, because the peristalsis of the bowels is so often influenced by psychic factors in ordinary life. Thus, fear will often cause diarrhoea, whilst depressive conditions, such as melancholia, very often bring about constipation. Constipation is largely a question of habit in every-day life. If people were taught to persevere regularly at fixed times, instead of flying to purgatives, they would be much better off. Bowel action is largely governed by the subconscious mind and it is for this reason that it is so largely a question of habit rather than of any pathological change in the alimentary canal.

It is for this reason that suggestion will often act more efficaciously than purgation. Very closely allied to this is the action of suggestion on the uterus, especially in the control of dysmenorrhoea and menorrhagia. It is especially suitable for cases where there is no obvious organic cause for the trouble. I have treated patients in whom these conditions have been cured and others who have been improved by suggestive treatment. Menstruation may in some cases be altered to a special day, which makes it probable that auto-suggestion is a factor in the onset of normal menstruation. I remember one girl in whom the pain accompanying her periods was so severe as to cause her to faint on many occasions. After one sitting the patient knew she was unwell only by finding the blood on her clothes. Previously she had to go to bed, but ever afterwards she was able to go about well and was quite free from pain.

There are many other cases in which suggestion has proved most beneficial, but in a paper like this it is not possible to refer to them all. I have had some good results in cases of more or less purely sexual nature and also when the ordinary conduct has not been what it should. The following is an example of the latter class:

Mr. X. was sent over to me by Dr. Chisholm Ross, from Sydney. He was a younger son of very wealthy parents. He seemed to have no idea of responsibility. He would drink heavily, take money and waste thousands of pounds on racing. He was abnormally addicted to doing things for ostentation, whilst he was unable to adapt himself to any business pursuit. He had been remonstrated with on numerous occasions, but to no avail. He was considered hopeless. His father and mother came over with him to

South Australia. I gave him twelve sittings with complete success. He changed from a heavy drinker to a teetotaler. He became moderate in horse racing, eventually giving it up. Then he applied himself seriously to the task of keeping going the immense business his father had built up. It is now several years since I treated him and he has changed from what is popularly called a "waster" to a successful, conscientious business man.

I have had similar cases which were also successful. I mention this last case to show how wide the application of suggestion is, which may be extended to both the physical and psychic activity of mankind. In conclusion, I would like to say that I am glad to see that the interest in practical psychology and psycho-therapy continues to grow in Australia and in the other parts of the world, and that practitioners are beginning to realize that it is not sufficient to know in a vague manner that man is half physical and half psychic. Both must be treated if the profession is to render the best services to humanity.

THE MEDICAL BENEVOLENT ASSOCIATION OF SOUTH AUSTRALIA.¹

By SIR J. C. VERCO, M.D., F.R.C.S.,
Adelaide.

THE COUNCIL very kindly granted the Trustees of the Medical Benevolent Association of South Australia permission to speak about its affairs at the last meeting of the Branch and entered on its Agenda Paper a notice of an address to be delivered by the Chairman of Trustees on the subject. But on that evening the weather was extremely hot and the other business lengthy and important, hence the subject was postponed for a month.

Since then the Annual Meeting of the Medical Benevolent Association has been held. We regret to report the absence of its Chairman to-night, owing to his visit to England, and of its Secretary, who is away on a month's holiday. But the other Trustee, Dr. A. A. Lendon, has consented to bring the matter before the profession. The Trustees wish first to convey to the Council their thanks for the opportunity now afforded him of doing this.

The method adopted to-night seemed to the Trustees the quickest, easiest and cheapest method of bringing the claims of the Medical Benevolent Association under the notice of the profession in our State.

This benevolent association in South Australia is doubtless known to all of you. It has been in existence since 1881, before many of you were born. Is it possible there are some members of our Branch who have never devoted anything to its funds? If so, it is probably as much a reflection upon its Trustees in not bringing its claims sufficiently often and forcibly before you, as upon the members of the medical profession in not showing a practical and liberal interest in it.

As an immediate result of the notice in the last Agenda Paper of the South Australian Branch of

¹ Written for a meeting of the South Australian Branch of the British Medical Association on March 29, 1923.

the British Medical Association a country member sent in a donation of five guineas and at our Annual Meeting held this month one of those present promised to become a life member by paying ten pounds and another to give twenty-five pounds a year for the next ten years. At the last meeting of the Branch a member expressed himself as quite prepared to pay a guinea a year at least. Now you are acquainted with its existence and its needs, the Trustees are sure you will be quick to help.

History of the Movement.

The origin of the Association is interesting. Before the South Australian Branch of the British Medical Association was established there existed a somewhat similar body named the South Australian Medical Association. In 1881 in the Board Room of the Adelaide Hospital the practitioners who constituted that Association, decided to wind it up. They had nearly two hundred pounds in the bank. And to their honour be it said, instead of squandering it in various ways as they might have done, they founded the Medical Benevolent Association and transferred all their capital to its three Trustees, Dr. Clindening, Dr. Paterson and Dr. Wyld.

Dr. Clindening was the grandfather of Dr. Gilbert, on whom his mantle has fallen and who has received a double portion of his spirit, for he is not only a Trustee, but the Secretary as well. Dr. Paterson was the uncle of our clever and genial member, Dr. Helen Mayo, and Dr. Wyld was a North Adelaide practitioner who was regarded as specially clever in eye diseases and whose practice was bought by Dr. Melville Jay, the father of our young ophthalmologist, Dr. Jay.

The Trustees to-day are Sir Jos. Verco (Chairman), Dr. Gilbert (Secretary) and Dr. A. A. London and the invested capital totals £1,080 19s. 3d..

The Object.

The object of the Association is: "To assist medical practitioners practising or who have practised in the State of South Australia or the distressed widows or families of such practitioners (a) by money advances, (b) by maintaining or educating the children of such practitioners.

Perhaps some will say: "Well, we have never heard of any practitioners or their widows or families who have been so assisted." Probably not. Yet many have been. And this demonstrates one desirable feature in its working. The fund is operated by the three Trustees and, as a rule, they alone know who receive its benefits. Were this published, it would probably aid not so many, because publicity would make some shy of applying. But any member of the Association can be supplied with desired information and can recommend for assistance anyone known to him to be in need of relief.

At the Annual Meeting, held always in March, after due notification, all may learn the financial position of the Association, what it has done through the year and what it proposes to do during the coming year.

Sources of Income.

Its sources of income are:

- (i.) The membership subscription of ten shillings a year. When founded it was one guinea. Money was in those days much dearer and harder to get; so that those venerable practitioners were more liberal than we are.
- (ii.) The life membership subscription of £10, of which very few have been received.
- (iii.) Direct donations to our capital fund.
- (iv.) Special donations for the assistance of special cases of need upon special appeal.
- (v.) Interest on our invested capital.

By our Articles of Incorporation the Trustees are allowed to spend in any one year:

- (a) The ordinary membership subscriptions for the current year, but not any life membership subscriptions; these have to be added to capital.
- (b) Any special donations for special cases.
- (c) The interest for the year on invested capital.
- (d) If in any year the receipts should exceed expenditure, twenty pounds of that excess may be transferred to the current account of the following year.

Last year the membership contributions totalled £6 13s. 9d., say £7, which would represent subscriptions at ten shillings a year from fourteen members. There are two hundred and ninety-one members on the roll of our Branch of the British Medical Association, so that more than 270 odd did not contribute anything. Surely some of the two hundred and seventy-seven are able and will be ready to give this year!

Our income from invested capital capital was £47 16s. 7d.. Our expenditure was £43 in relief and £2 11s. 6d. in legal expenses.

During the coming year the interest on investments will probably be £45 2s.. From last year's surplus income £20 can be spent this year and if the membership subscriptions this year are no greater than last year, namely, seven pounds, our total receipts would be only about £72.

Happily, since the notice on the Agenda Paper of February some donations and promises and subscriptions have come in, as already indicated.

Disbursements.

Last year one pound a week was given to an invalid lady, the elderly daughter of a deceased practitioner, for forty-three weeks. It is desired to continue the same weekly aid this year.

To another, a family bereaved of husband and father, it is desired to give at least a pound a week throughout the year.

When the Annual Meeting was held last month the available money for distribution this year was only about £72 and the Trustees were proposing to reduce the gift to the invalid lady of last year to £36 instead of a proposed £52 and to give only £36 to the new applicants. But at that meeting, owing to the generosity of some who attended it, this amount could be increased, but there is still not enough in view to warrant the promise of one pound a week to each. The Trustees, therefore, earnestly request the members of the profession to put down

their names as regular subscribers of ten shillings or more and so allow them to deal with the cases which come before them in a manner worthy of our profession.

Those of you who are young, would find it a profitable investment to pay ten pounds and so become life members, which would obviate the necessity of sending in annual subscriptions and prevent the possibility of forgetting to subscribe.

Misfortune or death may overtake any of us and those dependent on us may be left in straitened circumstances or even in actual want. As a thank offering that we are well and able to provide for our own, may we show ourselves ready to help those who are not so favoured.

Lindsay Gordon has written:

Life is mostly froth and bubble.

Two things stand like stone:

Kindness in another's trouble,

Courage in our own.

In our own personal difficulties may each one display the courage of a man and in the difficulties of our fellows the kindness of a woman.

Whatever we give to the needy we cannot lose, we shall not miss and we will not regret.

GLYCOSURIA.*

By A. E. MILLS, M.B. (SYDNEY),

Professor of Medicine, University of Sydney.

The Nature of Glycosuria.

THE term glycosuria simply means that glucose is present in the urine. Glucose is present in the urine of normal individuals, but only in very small quantities, which are not recognizable by the ordinary tests, *exempli gratia*, Fehling's, Benedict's. We may then strictly speak of a physiological glycosuria as we do of a physiological albuminuria. But when the glucose is in such an amount in the urine that it can be recognized by the ordinary clinical tests, that is, in a greater amount than is present in the urine of normal individuals with the same diet, then we may rightly speak of it as pathological glycosuria. It is now, however, firmly established that excess of sugar in the blood precedes the passage of glucose into the urine. In other words, before there is glycosuria there exists hyperglycæmia. What is meant by hyperglycæmia? To answer this we must have a knowledge of the concentration of glucose in the blood found in normal individuals. We must bear in mind that the amount of glucose in the blood varies very slightly; its constancy is very remarkable. Before breakfast glucose in the blood varies from 0.07% to 0.1%. After a meal rich in carbo-hydrate it rises to 0.10% to 0.15%. Two or three hours after a meal it reaches fasting level. If a normal person fasts for some time, the glucose in the blood remains at a level of about 0.07% from hour to hour.

This constancy of the blood sugar concentration depends upon many factors. These may be grouped

as those that prevent the blood sugar rising above normal levels and those that prevent the blood sugar sinking below normal levels. In the first group we have the conversion of glucose into glycogen, the burning up of glucose by the cells of the body for energy and the conversion of glucose into fat. In the second group we have in the first place the mobilization of glycogen in the liver and muscles and its conversion into glucose and in the second place an increase in protein metabolism, with increased formation of amino acids and the conversion of some of them into glucose.

The sugar from the intestinal canal is straightway absorbed and passes into the blood of the portal vein. In its passage through the liver the sugar—glucose—is withdrawn from the blood by the liver cells and converted into glycogen. When the absorption of sugar from the intestinal canal ceases, very little sugar exists in the blood of the portal vein. A reverse process now occurs. Some of the glycogen is converted into glucose, which passes into the blood of the hepatic veins. A very delicate relationship exists between the glucose concentration of the portal blood, the glycogen content of the liver and the glucose concentration in the blood of the hepatic vessels. The liver then, through its glycogenetic function, regulates the sugar content of the blood. The muscles begin to use sugar as soon as its absorption from the intestine begins. The body cells burn glucose more easily than any other food-stuff. When glucose is abundant, the combustion of fat almost ceases and the combustion of protein is reduced to a minimum. Glucose is burnt to carbon dioxide and water according to the following equation: $C_6H_{12}O_6 + 6 O_2 = 6 CO_2 + 6 H_2O$. This indicates that to burn glucose a certain volume of oxygen is required and for every volume of oxygen used a corresponding volume of carbon dioxide is given off. This relationship or ratio is known as the respiratory quotient. In this

CO_2
case — is equal to 1. In the burning of no other O_2

food-stuff is the quotient 1. For fat the quotient is 0.7. For protein the quotient is 0.8.

In normal persons four outlets exist which prevent the sugar accumulating in the blood in excessive amounts. These are (i.) the oxidation of glucose, (ii.) the formation of glucose into fat, (iii.) the conversion of glucose into glycogen, (iv.) the passage of glucose into urine. Normally the glucose concentration rarely exceeds 0.12% or 0.13%. We can take carbo-hydrates in the form of starch in great quantities, because their digestion and their rate of absorption is slow and we do not get an accumulation of sugar beyond 0.12% or 0.13%. If we ingest a large amount of carbo-hydrate in the form of glucose it is absorbed with great rapidity. Glucose enters the blood at a greater rate than can be dealt with by the means of regulation, *id est*, utilization, glycogen formation and fat formation are not sufficient to remove it all. It accumulates in the blood, the glucose concentration of the blood is raised and hyperglycæmia results. This may lead to glycosuria, but not necessarily.

* Being a lecture delivered at the Post-Graduate Course in Medicine in Sydney, January, 1923.

The height of concentration of blood sugar at which the kidneys excrete glucose differs in different individuals. This concentration level is known as the kidney threshold for sugar. In a few individuals the concentration of sugar in the blood before glycosuria appears in a recognizable amount may be only 0.08%. In some patients suffering from chronic nephritis the blood sugar may reach 0.26% before glycosuria results. The great majority of normal persons excrete glucose in urine in detectable quantities when the concentration of blood sugar is more than 0.15% to 0.16%. These facts show that the kidneys vary in their function with regard to the excretion of glucose as to other substances. When glycosuria results from too rapid absorption of glucose from the alimentary canal, it is called alimentary glycosuria. In such conditions the tolerance of the body for glucose has been exceeded. Glucose or carbo-hydrate tolerance is a term we must understand.

Seeing that glycosuria is the result of hyperglycæmia and that the kidney threshold for glucose excretion varies, we must get rid of this variable factor if we are to define what is meant by carbo-hydrate tolerance. We may define it as that amount of carbo-hydrate (glucose) which an individual can ingest without developing hyperglycæmia; it is then a test for the power and rapidity the individual possesses for the conversion of glucose into glycogen and into fat and for its combustion. If, for example, one hundred grammes of glucose dissolved in four hundred cubic centimetres of water to which the juice of one or one and a half lemons has been added, be given before breakfast to healthy persons it will be found that the blood sugar, which while fasting was present in a concentration of 0.07% or 0.1%, reaches 0.15% one hour after taking the glucose. Two hours later the blood sugar will be sinking to the normal level. Such a degree of hyperglycæmia is within the range of physiological variation. Now it may be asked if we have not the means of estimating the blood sugar, can we not estimate carbo-hydrate tolerance in any other way? Well, assuming that the kidney threshold for sugar is normal and that the hyperglycæmia may lead to glycosuria, if sugar can be found in the urine after the test meal of glucose, it may be definitely assumed that hyperglycæmia has been brought about and that the individual's carbo-hydrate tolerance is lower than normal.

It will help us to understand the subject of glycosuria better if we have some knowledge of the influences controlling glycogenesis, glycogenolysis and glycolysis. Our knowledge of these metabolic processes has been largely the result of experiment. We have first Claude Bernard's epoch-making discovery that puncture of the medulla led to glycosuria. Repetition of this experiment at a later date showed that the glycosuria was accompanied by hyperglycæmia and that the intensity of the reaction after puncture was dependent upon the nutrition of the animal, *id est*, the amount of the glycogen in the liver. Blair found that adrenalin injected into animals caused glycosuria, that this was preceded by hyperglycæmia and that repeated in-

jections of adrenalin into animals led to the complete discharge of glycogen from the liver. He also found that stimulation of the left great splanchnic nerve led to hyperglycæmia and that this stimulation resulted in the appearance of adrenalin in definite recognizable amounts in the blood. He showed that if the adrenals of rabbits be removed neither hyperglycæmia nor glycosuria resulted from puncture of the medulla and that if the sympathetic ganglia are put out of action by nicotine neither hyperglycæmia nor glycosuria follows puncture.

From all this it seems that glycosuria from puncture and from adrenalin act in the same way by stimulating the liver through the sympathetic nervous system. It has been established that every gland of internal secretion which has the power to stimulate the sympathetic, possesses power during a state of hyperactivity to cause glycogenolysis, leading to hyperglycæmia and glycosuria. But we must remember that this form of glycosuria is not due to the inability of the animal to use, *id est*, to burn up, sugar. In hyperthyroidism there is lowered carbo-hydrate tolerance, *id est*, hyperglycæmia, and this may lead to glycosuria. Cases have been recorded in which glycosuria resulted after taking thyroid gland extract in excessive amounts. One instance has been reported in which true exophthalmic goitre with glycosuria developed in a very fat person who took one thousand thyroid tablets in five weeks. In hypothyroidism, *exempli gratia*, myxœdema, there is increased tolerance for carbo-hydrate. The pituitary gland, like the thyroid, when in a state of hyperactivity affects carbo-hydrate metabolism. Borchardt found glycosuria in 40% of his patients suffering from acromegaly. We may say then that the pituitary extracts and adrenalin extracts and thyroid extracts act in a similar way. All have the power to stimulate the sympathetic and to produce hyperglycæmia and glycosuria. Any difference in their influence on carbo-hydrate metabolism is a difference of degree. The effect of each is to interfere with glycogen formation and its mobilization.

The influence of the pancreas in the production of hyperglycæmia and glycosuria is a different story. In 1889 von Mering and Minkowski made the great discovery that removal of the pancreas from an animal produced glycosuria and other symptoms of diabetes found in man. Later it was found that hyperglycæmia accompanied the glycosuria. It was also discovered that the glycosuria persisted when the food consisted of protein only and carbo-hydrate was entirely eliminated from the diet and that the sugar in the urine had a definite relation to the nitrogen excreted. Now nitrogen can only come from the breakdown of protein. Therefore the conclusion is that in depancreatized dogs fed on protein alone the glycosuria comes from the breakdown of protein, as does the nitrogen. No other endocrine gland seems to have the same effect on carbo-hydrate metabolism as the pancreas. The removal of the adrenal or the pituitary or the thyroid glands does not lead to glycosuria. Only in states of hyperactivity or as a result of excessive use of the extracts of these glands does glycosuria result. On

the other hand, removal of the pancreas produces glycosuria and the other symptoms of diabetes. Removal of a portion of the pancreas does not necessarily produce glycosuria; provided a certain amount of the pancreas is left no glycosuria results. In this respect the pancreas is like other organs of the body. The kidneys can and do perform their functions when much of their tissue has disappeared, as is seen in instances of interstitial nephritis. The liver is able apparently to functionate well, even when in a state of marked cirrhosis. In other words, as has been said, we have a large "factor of safety" in our glands and viscera.

It has been definitely established that four-fifths of the pancreas may be removed without producing diabetes. Whether glycosuria will develop after partial extirpation of the pancreas will depend upon the amount of pancreas that is left behind. Allen has shown that if less than one-tenth of the gland is left, the animal develops diabetes. If a little more is left, the animal may not have glycosuria if fed on protein, but if carbo-hydrate be given glycosuria will surely develop and then, even if the diet be restricted to protein, the diabetic state will continue.

There is now very definite and convincing evidence which points to the fact that the influence of the pancreas on carbo-hydrate metabolism is dependent upon an internal secretion or hormone. Let me quote the following experiments: (i.) If the pancreatic duct be tied, necrosis of the ordinary gland tissue of the pancreas follows, but the islets of Langerhans, some or all of them, remain unaffected and glycosuria does not follow. (ii.) Removal of the pancreas is not followed by glycosuria if a part of the pancreas is transplanted subcutaneously and the engrafted part survives. (iii.) If cross circulation of the blood be established between two dogs, "A" and "B," even if the pancreas of dog "B" be removed no glycosuria results in dog "B." (iv.) The pancreas has been removed from animals in the later stages of pregnancy. This was not followed by glycosuria or at rate only a slight glycosuria resulted; but after the birth of the puppies the mother became affected with true diabetes. (v.) In a depancreatized dog the glycosuria disappeared for a time after being transfused with the blood of a normal dog.

Can there be any doubt then that the pancreas supplies something—an internal secretion or hormone—quite apart from its external secretion, *id est*, the pancreatic juice, which enables the body to burn up glucose and to convert glucose into glycogen. In the absence of the pancreas this hormone is wanting and these two functions of the pancreas disappear, the body now has no power to oxidize glucose, nor can it convert glucose into glycogen. This results in hyperglycæmia and glycosuria. As Woodyatt quaintly puts it: "The glucose molecule in depancreatized animals and in severe cases of diabetes, figuratively speaking, enters into the chemical reactions of the body like so many glass beads; liberated in the body or introduced from without they rattle about until they fall out through the kidneys." This defect is present to a greater or

less extent in every case of diabetes. To a greater or less extent, for diabetes is a disorder of varying severity—mild, severe and fatal. So far we have dealt in a general way with the physiological processes that lead to glycosuria.

Sources of Glucose Supply.

We have seen that glycosuria is a result of an excessive amount of glucose in the blood, *id est*, hyperglycæmia. We have briefly referred to the influences bringing this about. We may next profitably consider the sources of glucose supply. It has been definitely determined that all carbo-hydrates (starches and sugars) that can be made use of by the normal animal are completely converted into glucose by the digestive and other metabolic processes of the animal body. In the condition of diabetes the various utilizable starches and sugars are readily converted into glucose. The diabetic has not lost the power of converting carbo-hydrates into glucose, but he has lost the power to a greater or less extent of burning or otherwise using the glucose so formed. Now the glucose so formed may be stored as glycogen in the liver and muscles and this in turn yields glucose and glucose only; glucose is the natural blood sugar. All investigations point to the fact that the body burns glucose and cannot burn directly any great amount of any other carbo-hydrate. The diabetic also has the power to assimilate the various carbo-hydrates. His defect is that he cannot oxidize the glucose molecule. But carbo-hydrates are by no means the only source of glucose.

Protein yields glucose in large quantities. In patients suffering from severe diabetes with glycosuria, if protein be given beyond a certain amount the glycosuria will increase. In dogs that have been completely phlorhizinized, protein feeding will increase the glycosuria. Lusk has shown in his experiments both in phlorhizinized dogs and in *diabetes mellitus* that the ingestion of one hundred grammes of protein leads to 3.65 grammes of extra glucose for every gramme of extra nitrogen excreted in the urine. Now one hundred grammes of protein yield about sixteen grammes of nitrogen. This means that 16×3.65 , or fifty-eight grammes of glucose, result from the breaking down of one hundred grammes of protein. In other words, one hundred grammes of protein may produce fifty-eight grammes of glucose. The ratio of 3.65 glucose to 1 nitrogen is not absolute, but so far it is the one best established. But the fact that I wish to emphasize is that protein may be a great source of glucose supply.

Now, emaciation is a striking feature in many patients with diabetes and the loss of weight will continue and the glycosuria will persist, even though carbo-hydrate be withheld from the diet, if much protein be given in the food. A normal person may live and work on meat and fat alone without there being any loss of weight. Some of the energy expended comes from sugar resulting from the breakdown of the protein of the food. In starvation the needs of the animal for the maintenance of life must be met. Part of these requirements comes from the glucose which is formed from the metabolism of

the protein of the tissues. This tissue breakdown leads to wasting. In diabetes wasting occurs because, though the food be sufficient, its glucose content cannot be oxidized; the fat and protein tissues are then called on. Their breakdown, while leading to the production of more sugar, also leads to more wasting of the body.

Lusk states that the Eskimo lives almost entirely on protein and fat. In times of plenty he may eat as much as four kilograms (nine pounds) of meat daily. He certainly gets a little carbo-hydrate from seaweed, the young shoots of angelica, mussels and from the glycogen contained in the skin of young whales, but all this yields little more than 8% of the calories of his food. Contrast this with the diet of a European in which carbo-hydrates form 67% of the calories of his food. It is true that the Eskimo eats nearly three times as much fat in his food as does the European, but what of the excessive amounts of protein that he consumes? It is broken down into amino acids—the building stones of the body as these fragments have been called. Some of these fragments produce large amounts of glucose, which provides a great part of the energy the Eskimo requires. But there is another source of glucose supply besides that which comes from carbo-hydrates and protein. It is well known that glycerol results from the breaking down of fats, that glycerol is capable of being entirely converted into glucose in phlorhizinized dogs and that it increases the glycosuria of the diabetic.

Woodyatt calculates that one hundred grammes of fat catabolized in the body may set free ten grammes of glycerol and so yield ten grammes of glucose. Thus we arrive at the conclusions that carbo-hydrates, proteins and fats all yield glucose, that the carbo-hydrates are completely converted into glucose and that 58% of the protein and 10% of the fat may be converted into glucose.

Acidosis.

It is common knowledge that acids accumulate in the blood and tissue fluids in various disorders of the body, *exempli gratia*, in nephritis, in diabetes, in starvation, in hyperemesis of pregnancy. In nephritis there is a failure on the part of the kidney to excrete those acids which are the products of normal metabolism. In diabetes, in starvation and in excessive and prolonged vomiting, which really is starvation, foreign acids are added to the blood. In all these conditions we may have the usual signs of acidosis because the excess of acids—normal or abnormal—produces the same effects, *id est*, the depletion of the stores of the bicarbonate of the blood, causing changes in the carbon dioxide of the alveolar air, in the reserve alkalinity of the body and in the acid excretion by the kidney. In diabetes and in starvation the acids which accumulate, are diacetic acid and β -oxybutyric acid. These acids are the result chiefly of a faulty metabolism of fat. For the complete combustion of fat in the animal body a certain amount of carbo-hydrate must be burned at the same time.

We have seen that fat is not so readily burned as sugar. It needs the fire of the burning sugar to

burn it completely. If the carbo-hydrates are not burned in sufficient quantities, the fats are incompletely burned. Intermediate products or products of incomplete combustion result. These products are diacetic acid and β -oxybutyric acid, acetone or ketone bodies. Such incomplete combustion occurs in diabetes and in starvation. In diabetes because there is a disability to burn up sugar and in starvation because there is a deficiency of sugar to be burned. In both conditions, however, the production of these acid bodies resulting in acidosis is due to the same fundamental cause, namely, an improper or ill-balanced metabolism of fat and carbo-hydrate. But it must also be borne in mind that protein may yield these same ketone bodies, certainly not to the same amount, when there is a defective combustion of carbo-hydrates. In the case of a normal individual who abstains entirely from food, it is found that for the first few days none of these acid bodies appear in the urine. The stores of glycogen in the liver and muscles are sufficient for the complete combustion of fat. The proper relation between carbo-hydrate and fat combustion has been maintained. Later on these acid bodies appear, for the glycogen store has now been depleted and the fat cannot now be thoroughly burned. If the starvation is prolonged, the fat supplies as well as the carbo-hydrate supplies are used up, the proteins alone remain for the maintenance of life. During this period the acid bodies will diminish for the reason that in the metabolism of protein, as we have already mentioned, about one-half of the protein is converted into glucose which by combustion burns up or helps to burn up the other half.

For these reasons in very fat persons during starvation marked acidosis will result, but in very lean persons during starvation there will not be marked acidosis, because there is little fat to be burned. The organism must depend chiefly upon its protein for fuel and the protein, as we have seen, provides its own sugar during metabolism for its more or less complete combustion.

In diabetes the same principles apply. The power to use carbo-hydrates is wanting. There must be a balance between the burning of fats and carbo-hydrates and, as this balance is disturbed, the diabetic can only completely oxidize a smaller amount of fat.

To state the case briefly, an amount of fat which a healthy body can burn up, is imperfectly burned by the diabetic and acidosis results.

RANDOM OBSERVATIONS ON MITE INFESTATIONS OF MAN.

By R. W. CILENTO, M.D., B.S. (ADEL.), D.T.M. & H. (LOND.),
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THE infestation of the human host by various forms of mites is neither rare nor unusual and the species implicated are numerous. In spite, however, of the great contributions to our knowledge of insect pests in Australia which have been made by

J. Burton Cleland, T. Harvey Johnston, S. J. Johnston, Georgina Sweet, Ferguson, Breinl, Priestley, Nicoll and others, the closest scrutiny of the literature reveals only a few scattered observations regarding mite infestation in Australia.

Cases of skin irritation definitely traceable to *Sarcoptes hominis*, *Laelaps agilis* (parasitic upon *Mus decumanus*), *Leptus* species, *Dermanyssus avium* (the common fowl mite) and *Liponyssus bursa*, Berlese (the tropical fowl mite) have all been recorded and gamasids from hens, starlings, English sparrows (probably of one or the other of the last two types mentioned above) have been reported in several instances. In one instance the pest was such that it was necessary to remove the roof of a church and to clear out the nests of the birds which had established themselves amongst the rafters, before the congregation could assemble in comfort.

"Grocer's itch," "grain itch" and "copra itch" are other well-known varieties of skin irritation, the last being especially familiar to workers in New Guinea, while the so-called "red spider," *Leptus* (*Trombicula*?) species, also mentioned by Cleland, is found in many parts of Queensland and New Guinea and produces the maddening irritation known as "scrub-itch."

The attention of the writer was directed towards the question of mite infestation of the human host by the study of the affinities existing between the epidemic glandular fever of Queensland and Japanese river fever, the fever conveyed in Japan by the larval mite, *Microtrombidium* (*Trombicula*?) *akamushi*, which so closely resembles the local *Leptus* species.

The search amongst the literature resulting in a meagre return, attention was directed towards collections made from time to time at previous dates by Mr. Gerald F. Hill, the entomologist to the Institute of Tropical Medicine. It was found that mites of the most varying kinds and species parasitic on many varying hosts were extremely prevalent in the tropical localities of Australia and its dependencies.

Thus a large unidentified mite almost the size of a small cattle tick was discovered upon one of the *Paussidae*. A small red tick was found on a specimen of *Tabanus avidus* from Magnetic Island, near Townsville, North Queensland. Three separate species (unidentified) were obtained from the bodies of queen termites collected in northern Australia. Specimens of *Mansonioides uniformis* from Biot, in Papua, were found to be swarming with a variety of little white mite, one or more species were found in enormous numbers in cow-dung and besides the form which causes "red spider" itch or "scrub itch" (and possibly the "black soil itch" of Camooweal, West Queensland), numerous free-living species were found attacking tabanid larvæ in breeding jars.

By a curious coincidence, during the course of these observations the writer disturbed a pigeon's nest on the eaves immediately above a window of one of the laboratories at the Australian Institute of Tropical Medicine and was showered with specimens of the tropical fowl mite, *Liponyssus bursa*, while about the same time many specimens of the

same mite engorged with blood were sent in from patients in Townsville by Dr. T. Gordon Ross. These cases are interesting as being the first reported from tropical Australia so far as can be ascertained and Dr. Ross has kindly attached the following histories:

CASE I.—Mr. A.B., aged forty-four years, stated that on getting up one morning he felt a slight itching around his ears and neck. On rubbing his hand over his neck he found a few small insects and then found a few more on his pillow. He did not feel anything further during the day, but next day on examining his pillow again he saw a number of insects and got a few on his face and shoulders. This occurred each morning for several days. There was no irritation or discomfort during the rest of the day. On examination no rash was visible on the patient's skin, no marks of insect bites, but some of the insects collected by the patient were found to contain blood. The patient used a weak "Lysol" lotion for sponging every morning and soaked his pillow slips in lotion when insects were discovered. After a few days no more were seen.

CASE II.—Mr. C.D., aged forty-five years, complained of a severe itching on the limbs and trunk which he had had for several weeks. On examination a rash was found which appeared to be identical with scabies, but there were not many pustules and the rash was not present between the fingers. He stated that he was in the habit of handling fowls a good deal and often on putting his hand into the nests would get his hand and forearm covered with little black insects. These, however, usually left his body very quickly and he was not apparently troubled by them when away from the fowls. He was asked to report again and bring some of the "fowl lice" for examination. Up to the present he has not done so.

Liponyssus bursa (Berlese) is widely distributed. It has been reported from the following localities among others: North and South Nigeria, Zanzibar, Nyassaland, Zululand, Conoro Island, Mauritius, India, China, United States of America, Bahamas, Trinidad, South America and Australia.

In Australia Cleland has reported instances from Cremorne, Sydney, New South Wales, and from Port Pirie, South Australia. The symptoms in these instances were those of intense irritation and rash.

Cases have also been reported by Howell from Achmednagar, India, where a lady suffered from a very bad irritation of the skin, alleged to be caused by the mite. It raised small red lumps with white tops. The appearance suggested that the insect burrowed and the irritation was intense even for days after the bite.

While it is not suggested that the mite in question or any other tropical Australian variety is necessarily the disseminator of any disease, the facts that it will attack man and that Hirst claims it to be the possible transmitter of spirochaetosis in fowls render its presence in an area where there is endemic a disease closely resembling the mite-borne river fever of Japan, a matter of interest. Moreover, the very great prevalence of other mites of many varieties and the frequency of rashes and fevers of an unknown origin in some of the areas where they are prevalent, indicate that an investigation into the Australian tropical acarines is called for and might prove of importance.

Summary.

1. Investigation, initiated in the search for possible affinities between the epidemic glandular fever of Queensland and Japanese river fever, revealed

the presence in tropical Australia and its dependencies of a considerable number of varying species of mites.

2. During the course of this investigation two cases of infestation by *Liponyssus bursa* (Berlese) came to the notice of the writer. So far as can be learned, these cases which are here recorded are the first reported from these areas, though doubtless by no means the first seen.

3. The possibility of the presence of mite-borne diseases in tropical Australia suggests that a study of the local mites would be desirable and possibly important.

References.

- Cleland, J. B.: *The Australasian Medical Gazette*, September 14 and 21, 1912; reprinted in *The Journal of Tropical Medicine and Hygiene*, Volume XVI., page 43 et sequela.
- Thomas, D.: *The Australasian Medical Gazette*, December, 1884, page 65 et sequela.
- Hirst, S.: *Bulletin of Entomological Research*, Volume VI., 1915, pages 55 to 58.
- Berlese, Antonio: *Bullettino Società Entomologica Italiana*, Volume XX., 1888, page 208.
- Hirst, S.: *Annals and Magazine of Natural History*, Volume XVIII., 1916, pages 243 and 244.

Reports of Cases.

ANAPHYLAXIS WITH SUDDEN ONSET.

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THE following history is thought worthy of report on account of the severity of the symptoms and the rarity of the condition:

Clinical History.

J.S., male, *etatis* nineteen years, was admitted to the out-patient department of the Ipswich Hospital at 8.30 p.m. on April 3, 1923. While playing football he had received a cut over the left eyebrow five centimetres (two inches) long.

On examination no head, abdominal or chest injury was detected. His wound was cleansed, sutured and dressed. He was given anti-tetanic serum (1,500 units) after having denied ever receiving previous serum treatment. After three-quarters of an hour and apparently feeling perfectly normal he left the hospital in the company of a friend.

At 10.15 p.m. (ten minutes after leaving the hospital) he was again brought in by a police constable, who supposed him to be intoxicated. A few minutes after leaving he had felt cold and ill and had commenced to vomit, first stomach contents and later watery fluid.

On examination his face was seen to be drawn and pallid, his pupils were dilated and his eyes rolled upwards. The skin was cold, moist and clammy. He was pulseless and the heart beats could be heard but faintly on auscultation. In fact, he was absolutely collapsed; his mind was clear and he could talk quite rationally, but he thought that he was going to die.

He was put to bed, was surrounded with hot water bottles and was given a hypodermic injection of 0.003 gramme (one-twentieth of a grain) of strychnine. The foot of the bed was raised and saline solution was administered continuously *per rectum*. His temperature was 35.3° C.

(95.6° F.). His respirations numbered fourteen per minute. No head, chest or abdominal injury could be detected.

At 10.45 p.m. one cubic centimetre of pituitrin was given hypodermically and his skin became warmer. His eyelids were swollen and the vomiting continued. He complained greatly of thirst.

At 11.15 p.m. a second hypodermic injection of 0.003 gramme (one-twentieth of a grain) of strychnine was given.

At 12.30 a.m. he was seen by Dr. Trumpy and by this time was vomiting blood-stained fluid and a rash had developed on his limbs. At 1 a.m. 0.3 cubic centimetres of adrenalin (one in 1,000) were given and an hour later he was much improved. The temperature was 37.3° C. (99.7° F.), the pulse had improved in rate and volume, but was still uncountable.

At 2.15 a.m. one cubic centimetre of pituitrin was injected and from this stage he gradually improved.

At 8.30 a.m. on April 4, 1923, his rash had disappeared, vomiting had ceased, his temperature was 37.2° C. (98.6° F.), his pulse rate was 120 and his respirations numbered eighteen per minute. He had retained two and a quarter litres (four pints) of saline solution, which had been administered *per rectum*. He was given a fluid diet and made an uneventful recovery.

He was sitting up in bed on April 6, 1923, and on April 8, 1923, was taking a full diet. He was discharged on April 9, 1923.

At 4.30 p.m. on the same day, ten minutes after returning home, he began to develop pruritus of the limbs and body.

At 5.30 p.m. he was seen by Dr. Paterson, who found a well-marked rash present on the body and limbs, together with a swollen neck, stertorous breathing, cyanosis, oedema of the glottis and expectoration of a quantity of clear, frothy, colourless fluid.

He was re-admitted to hospital at 7.30 p.m. On examination the temperature was found to be normal, the pulse rate was 96 and the respirations numbered 24 per minute. Nothing abnormal was detected in the heart, abdomen or nervous system. Examination of the lungs showed moist sounds to be present all over the chest. A well-marked erythematous rash was present on the trunk and limbs, the eyelids were puffy, the throat was red and injected, the uvula was oedematous, while the neck was now no longer swollen and there was no difficulty in breathing. Sputum was now more scanty and the patient was feeling very ill. Calcium lactate was given every four hours in doses of one gramme (fifteen grains) and calamine lotion was applied externally.

At 10 p.m. he felt more comfortable, but complained of itching. A hypodermic injection of morphine of 0.01 gramme (one-sixth of a grain) was given.

On April 10, 1923, he complained of pain in the joints, especially the shoulder, knee and hip joints. These were swollen and contained fluid in slight amount. His recovery was rapid and uneventful. He was discharged on April 15, 1923.

Comment.

1. The patient denied having had any previous serum treatment, but after much questioning admitted to having had three injections of anti-influenza serum in 1918.

2. He developed serum sickness seven days after his injection on April 3, 1923, and this rather confirms the diagnosis of anaphylactic shock on April 3, 1923.

3. The anaphylactic shock coming on three-quarters of an hour after the injection is interesting.

4. Kolman says: "Serum sickness and anaphylactic shock are more prone to follow anti-tetanic serum than anti-diphtheritic serum on account of the former being less concentrated and due to the less efficient removal of toxic bodies."

5. Only forty cases of anaphylactic shock are reported following within a few minutes of injection of serum and are characterized by dyspnoea, prostration and death. In one series of cases 0.9% of the patients showed signs of anaphylactic shock within twenty-four hours.

A REPORT ON FOUR CASES OF TETANUS
IN CHILDREN.

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AND

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The complete recovery of these four children from tetanus after energetic treatment with anti-tetanic serum, is worthy of record. It is of especial interest in view of the comparatively short incubation period—in each instance seven days. This places them on the border-line between the acute variety of infection, which is characterized by a very short incubation period and is almost invariably fatal, and the more chronic type, in which a longer time has elapsed before the onset of symptoms. We would suggest that these successful results afford further evidence of the efficacy of large doses of the serum given early and particularly by the intra-theal route.

Clinical Histories.

A.P., *etatis* five years and eleven months, was admitted to hospital on October 25, 1922, with the history of having sustained a small wound of the left leg by puncture from a stick twelve days before. He complained of stiffness

the left leg and had a limp of five days' duration. He also had trismus for three days prior to admission. On examination there was found to be slight generalized rigidity, definite trismus and rigidity of the flexor muscles of the left leg and thigh. Mentally he was bright.

R.C., *etatis* thirteen years and six months, was admitted to hospital on October 31, 1922, with the history of having received a scalp wound following a dive into shallow water eleven days before. He complained of occipital headache, stiffness of the neck and dysphagia of four days' duration. Trismus had been present for two days. On examination marked generalized rigidity and trismus were found to be present. Mentally he was bright.

W.C.J., *etatis* nine years, was admitted to hospital on February 4, 1923, with the history of having injured the right foot eight days before. He complained of stiffness of the neck, pains in the back and trismus which had been present for the previous twenty-four hours. On examination definite generalized rigidity and trismus and definite rigidity of the muscles of the right leg were present.

G.B., *etatis* seven years, was admitted to hospital on February 24, 1923, with the history of having run a rabbit bone into the right foot ten days before. He complained of stiffness of the jaw of three days' duration. This was accompanied by painful spasms of the spinal and facial muscles. He had been given six thousand units of anti-tetanic serum intramuscularly before admission. On examination definite trismus and frequent generalized spasms were present. Mentally he was bright.

In all the typical "*risus sardonicus*" was very pronounced and in each instance serum treatment was commenced immediately after admission and continued as shown in the accompanying table:

Patient.	Incubation Period.	Duration of Trismus Before Treatment.	Day of Treatment.	Dosage of Serum in American Units.					Remarks.
				Intra-thecal.	Intra-venous.	Intra-muscular.	Subcutaneous.	Total.	
A.P. . .	7 days	3 days	First	13,000	—	7,500	—	20,500	Several short spasms
			Second	15,000	—	5,000	—	20,000	Several short spasms
			Third	10,000	10,000	10,000	—	30,000	Three short spasms
			Fourth	6,000	—	3,000	—	9,000	Two short spasms
			Fifth	—	—	—	3,000	3,000	Splinter removed from wound
			Sixth	—	—	—	3,000	3,000	No serum rash
			Total	44,000	10,000	25,500	6,000	85,000	Discharged on twentieth day
R.C. . .	7 days	2 days	First	15,000	10,000	10,000	—	35,000	Two short spasms
			Second	15,000	—	15,000	—	30,000	Several short spasms
			Third	15,000	—	15,000	—	30,000	Two short spasms
			Fourth	15,000	—	15,000	—	30,000	Cerebro-spinal fluid normal
			Fifth	—	—	—	3,000	3,000	Serum rash on ninth and
			Sixth	—	—	—	3,000	3,000	tenth days
			Total	60,000	10,000	55,000	6,000	131,000	Discharged on nineteenth day
W.C.J..	7 days	1 day	First	10,000	5,000	12,000	8,000	35,000	Four spasms; cerebro-spinal fluid normal
			Second	17,000	—	18,000	—	35,000	Several short spasms
			Third	17,000	—	18,000	—	35,000	Cerebro-spinal fluid "grossly purulent"; sterile on culture
			Fourth	16,000	—	19,000	—	35,000	Trismus less marked
			Fifth	—	—	10,000	—	10,000	Several short spasms
			Sixth	—	—	10,000	—	10,000	Serum rash on ninth and
			Seventh	—	—	5,000	—	5,000	eleventh days
Total	60,000	5,000	92,000	8,000	165,000	Discharged on twentieth day			
G.B. . .	7 days	3 days	First	20,000	8,000	8,000	—	36,000	Cerebro-spinal fluid clear and under increased pressure
			Second	8,000	—	5,000	—	13,000	Limited supply of serum
			Third	15,000	10,000	10,000	—	35,000	Spasms less severe
			Fourth	13,000	—	12,000	—	25,000	Fluid turbid but sterile
			Fifth	8,000	—	7,000	—	15,000	Very few spasms
			Sixth	—	—	10,000	5,000	15,000	
			Seventh	—	—	10,000	—	10,000	
Eleventh	—	—	5,000	—	5,000	Few spasms			
Total	64,000	18,000	67,000	5,000	154,000	Discharged on twenty-first day			

Lumbar puncture and intra-theclal administration of serum were always carried out under general anæsthesia. The concentrated anti-tetanic serum was used for intra-theclal injection.

Sedatives were given freely, the most effective being chlorotone, chloral and sodium bromide.

The cost of the serum, according to the list prices of the Commonwealth Serum Laboratories, exclusive of the discount allowed to the Hospital, was £61 10s. 6d. or £15 7s. 6d. per patient.

Summary.

1. These results emphasize the importance of the early administration of large doses of anti-tetanic serum. By intra-theclal and intravenous administration the serum is brought rapidly into the closest possible contact with the toxin. Intramuscular and subcutaneous injections provide a reservoir from which continuous absorption occurs.

2. Turbidity of the cerebro-spinal fluid is usually met with following the second or third intra-theclal injections and is due to the presence of a large number of polymorphonuclear leucocytes. Culture of the fluid is sterile. It is merely an aseptic reaction to the serum and need cause no alarm.

3. Definite decrease in frequency of the spasms cannot be expected during the first few days of treatment and the progress may be regarded as satisfactory if the spasms do not increase in severity.

Acknowledgements.

We are indebted to Dr. S. W. Ferguson, Dr. R. L. Forsyth and Dr. H. H. Turnbull for permission to publish the histories of these patients.

Reviews.

THE ADMINISTRATION OF ANÆSTHETICS.

THIS handbook of Dr. Hadfield¹ effectively fulfils his design of furnishing the medical and dental students, as well as young practitioners, with a means of supplementing their practical instruction with such theoretical and practical information as will enable them to give anæsthetics for all usual operations. He has effected it in such a manner as to emphasize essentials without having them obscured by the intrusion of less essential matter. Those seeking a more comprehensive grasp of the scientific principles involved are referred to larger text-books. Close attention has been given to the small but important manipulative details which he has proved of value in personal experience.

The chapters on general considerations and the signs and stages of anæsthesia are concise but sufficient; those on nitrous oxide and nitrous oxide with oxygen are more comprehensive, for the benefit of the dental students and those desirous of mastering the technique for administration in minor and major surgery. The subject of ether anæsthesia is well treated; due attention is given to Clover's or the closed method, the open method, as well as to the more recently introduced methods of administration by the rectal and the intra-tracheal routes and by the pre-warming method. The author gives preference to the ethyl chloride-ether sequence by means of Clover's apparatus and to McCardie's mitigated ether mixture for routine usage. Rather less importance than seems warranted is given to the use of morphine in minimal dosage with atropine in conjunction with ether. It is to be regretted that notice is given in text-books to such substances as "Ethanosal." The announcement by Cotton of his discovery that pure ether has no anæsthetic properties and that such as is possessed by commercial ether is due to im-

purities does not appear to have been confirmed in research by others and as the essential qualities in any general anæsthetic agent are so pronounced in "pure" ether as sold and, moreover, as the purest of ether hitherto made by others has been found to have anæsthetic qualities quite as active as the commercial article, it is unlikely to be confirmed. Exploitation of anæsthetists with empirical mixtures of ether with more or less of "certain ketones" based upon Cotton's claim savours of charlatany and should not be countenanced by notices in medical literature. The directions for administering chloroform and ethyl chloride are concise and adequate. Very useful articles, such as anæsthesia for children, anæsthesia for special operations and pathological conditions, spinal anæsthesia, the treatment of accidents and emergencies occurring to anæsthetized patients and on shock, complete the work. It is an excellent handbook and it brings this class of literature up to date.

OPHTHALMIC SURGERY.

LIKE many books written by Indian surgeons, there is a breeziness about Major Nesfield's "Ophthalmic Surgery" which is quite refreshing.¹ He states fearlessly his own opinions, propounds his own theories, adopts his own treatment and employs his own spelling. Scornful of etymology, our familiar pterygium become "tarigium." Excepting perhaps for the last count, we do not blame him. Why write a book at all if one cannot set out one's own views? The result is infinitely more entertaining and, as Punch remarks in another connexion, it won't poison us and does not bind us to vote for him. To understand fully the Indian author it is necessary to know the atmosphere of the Indian civil hospital, the rush of work, the crowd of patients, the rough-and-ready diagnosis, the quick decision and the immediate operation. Then, too, there is the prestige of the *Sahib*, the absence of discussion, the take-it-or-leave-it attitude and lastly to a large extent the irresponsibility and fatalism regarding results. It is something like playing auction bridge without stakes. In surroundings like these the Anglo-Indian finds his enormous material and unlimited opportunities; but there are dangers and pitfalls as well.

Frankly, in the present volume of one hundred and seventy-two pages the author has attempted too much. Within such a compass it is impossible to present adequately such a wide range of ophthalmological subjects as we find here introduced. It is equally impossible in criticism to do more than touch on a few important points. The intra-capsular extraction of cataract is warmly advocated and the author has introduced a modification of the Smith technique. This consists in the use of a ligature, a gold wire with a smooth, bent end, which is passed into the corneal incision under the iris below the point of rupture of the suspensory ligament. The extraction then easily follows. The author presents a good argument and claims greater success and safety with this manœuvre. For glaucoma we note that Elliot's trephining is the procedure described and recommended.

Sub-conjunctival injections are employed in the treatment of many conditions, such as corneal opacities, the drug recommended being "Phenolaine" on account of its antiseptic and analgesic properties. So far we have not been able to determine the composition of this drug; it is unfortunate that no light is thrown on the point either in the text or in a footnote.

In the operation for removal of a staphyloma as performed formerly at Amritsar, the sutures are inserted before the staphyloma is excised. This seems a distinct advantage.

We feel indebted to the writer for many a good hint and for many suggestions possibly unorthodox, but certainly stimulating. To give one example. Conical cornea, he says, is a form of glaucoma.

¹ "Practical Anæsthetics for the Student and General Practitioner," by Charles F. Hadfield, M.B.E., M.A., M.D. (Camb.); 1923. London: Baillière, Tindall & Cox; Demy 8vo., pp. x. + 244, with 32 figures in the text. Price: 7s. 6d. net.

¹ "Ophthalmic Surgery," by Major V. Nesfield, Major, I.M.S. (Retired), F.R.C.S. (England); 1922. London: H. K. Lewis & Company, Limited; Demy 8vo., pp. xii. + 172, with 22 illustrations. Price: 9s. net.

The Medical Journal of Australia

SATURDAY, MAY 19, 1923.

An Educational Problem.

MEDICINE is often spoken of as one of the liberal professions. Its practitioners are called doctor or learned, some as a right in virtue of a special degree granted by a university, some as a matter of courtesy and expediency, for the public has long since associated the calling of the medical practitioner with the title of doctor. In the British Empire medical practitioners are entitled to registration and official recognition if they have gained one of a very large number of degrees or diplomas, each of which has a different standard. From the professional point of view there is a lack of uniformity of equipment judged from the standpoint of the qualifying examination. In no other country is there so great a difference between the minimum and the maximum, with the possible exception of America.

It is but a natural consequence of this wide latitude of standards of professional knowledge that there should be among medical practitioners a very great difference in the level of general education. Under all conditions a considerable variation must exist in the general and special knowledge possessed by practitioners of medicine, for the personal equation and the individual aptitude and intellectual attainments are involved. But few persons will question the undesirability of a wide divergence of standards either for professional or general education. If a serious attempt were made in all parts of the Empire to introduce a fixed standard for qualifying examinations, not a minimum, the fixing of a uniform standard of matriculation or other entrance examination would follow as a matter of course. It must be understood that we are not suggesting that a standard for examinations implies uniformity in education. Unfortunately the examination test is still regarded as the best method of assaying the knowledge possessed by

the candidate. For this reason, uniformity would be more readily attained at present by the introduction of fixed standards for examinations than in any other manner.

The medical profession in all parts of the British Empire is becoming overcrowded. In Australia the overcrowding is not yet acute, but it is nevertheless evident. One of the remedies for overcrowding is to render it more difficult for boys and girls to enter the medical schools. Moreover, it is kinder and wiser to eliminate those whose intellectual equipment is not of a high order, at the outset than later. The preliminary training given to prospective medical students should be sufficiently extensive and adequate to guarantee to all those completing the course a smooth passage into the medical profession, provided that they apply themselves diligently and earnestly to their professional studies. At present the education of the young person destined to enter the medical profession in no way differs from that of any other person. There is no provision for extra or special classical, mathematical or scientific training. In some of the great public schools in Great Britain there are special classes for senior boys wishing to enter the British Army as commissioned officers. Boys who select the Navy, are sent to the recognized special schools.

Those who are to become doctors, are offered the same opportunities and the same impediments as those whose future lies in commercial pursuits. Apart from the fact that medicine is a science and that its practitioners are supposed to be learned, the very essence of the doctor's daily work renders it imperative that he should be a man of varied attainments, capable of understanding human nature in its infinite varieties and of being the counsellor, friend and referee of his patients. In addition to intelligence, he must have an ample fund of knowledge at his disposal and competence to weigh facts and to judge their significance.

There is a tendency to pamper youth by endeavouring to lessen his burden during his school and student years. Even the brilliant boy or girl will find it difficult to equip himself or herself satisfactorily for his responsible life. Surely it is a

mistake to lessen the burden, when this necessarily means a sacrifice of effective preparation for that strenuous duty. Moreover, it is difficult to exclude a single ordinary school subject from the list. He must be a master of his own language. He will need it every day of his life. The use of words is of paramount importance and this is the first essential of good education. Every medical practitioner should be able to write and speak his own language fluently, grammatically and easily. We would urge that no student should be allowed to enter a medical school unless he can produce evidence that he can write and speak correctly and well. By well is meant that the spoken or written sentence should convey the intended meaning. At times the careless choice of words leads to a meaning which was not intended.

In the second place, the student should have a knowledge of literature. This entails the earnest study of the writings of the masters, not only of our own, but also of two classical tongues, namely, Latin and Greek, and of at least two modern languages. For the medical practitioner French and German are indispensable. Italian is useful and highly desirable on account of the extraordinary beauty of its classical literature. Latin and Greek should not be taught as dead languages. This method is soul-killing, tedious and ineffective. As mental exercises Latin and Greek, learned as modern languages are learned, are stimulating, little exhausting and unequalled. The student obtains a love for the masterpieces of the great poets and historians of ancient Greece and Rome. The third subject which enters the scholastic curriculum, is mathematics. It is obvious that no relief can be sanctioned in this direction, for modern medicine demands an ever-increasing application of higher mathematics and physics to its special problems. Then come subjects such as history and geography; every student of local or general politics will appreciate the necessity of a good working knowledge of the world and the important events in each section in every age. There remain sciences and arts. A full appreciation of music and the reproductive arts is impossible without some special study. The history of music and that of painting and sculpture are so interesting and fascinating that this subject

could be taken almost as a recreation. Life would be unbeautiful indeed if men and women were unable to appreciate the soothing influence of good music or the elevating effect of a fine painting. Finally, there is the difficult question of the pure sciences. We have advocated a sound preliminary training in the elements of chemistry, physics and zoology. Once the student enters the medical school he should be prepared to apply all the fresh knowledge he acquires to some problem in medicine. It is economical to require the schoolboy or school-girl to have a general idea of principles and some knowledge of simple chemical equations before the school days are closed. Moreover, if the school training is to serve as a test of the fitness of the student for the profession of medicine, the inclusion of elementary science becomes imperative. Otherwise, how is the teacher to judge whether the student has any scientific ability? It will thus be seen that the preliminary education for those desiring to become medical practitioners must be extended and made more advanced than it is. The difference should be recognized by the establishment of special classes in the senior school and the appointment of special teachers, who would have to take cognizance of the requirements of the learned profession of medicine.

Current Comment.

THE DISTRIBUTION OF METABOLITES IN THE BODY.

THE various food-stuffs which are taken into the body are first submitted to the preparatory action of enzymes. They are thus rendered fit for absorption. Before absorbed products can be utilized in the metabolism of the body, dissociation takes place by a process of oxidation in order that the products may take part in the vital chemical reactions. It has been assumed that the vital processes are subject to standard conditions of temperature, hydrogen ion concentration and general chemical balance. The quantitative estimation of the products of metabolism has been studied and the knowledge gained has been of much use in the clinical aspect of disease. Sugar, urea and creatinin are the products that have been investigated more particularly in this regard.

It is known that the concentration of sugar in the blood of normal persons tends to remain at a fixed level. This level is disturbed after the ingestion of sugar and the concentration in the blood immediately rises. In a few hours it falls again to

the original fixed level. Ultimately the sugar is claimed by the tissues and rapidly burned to carbon dioxide. The ability of the blood and the tissues to hold glucose has been studied in various ways. At present there is no means of ascertaining the amount of glycogen from which the glucose is drawn. It is obviously a matter of importance to discover whether any mechanism exists whereby the concentration of the sugar deposited from the blood in the tissues in health tends to remain at a fixed level. If such a mechanism were found to exist, it would be necessary to ascertain whether a parallelism existed between the rate of dissociation of the sugar in the tissues and the amount available. Further consideration would have to be given to the disturbances of such a mechanism in pathological conditions associated with an alteration of metabolic processes.

In the dissociation of protein the conditions are even more complicated. It must be remembered that, while in the ordinary course of events peptones are oxidized to the amino-acids and many other complex chemical bodies, under certain conditions glucose may be formed from the partial oxidation of protein. The difficulties involved in following the processes of metabolism by studying the concentration of urea and creatinin and the nitrogen balance are considerable, since there are at present no methods available of estimating the actual values of the nitrogenous bodies in the tissues or the compounds of urea, creatinin or uric acid.

Many researches have been conducted with a view to the establishment of the sugar and urea constant of the blood. The curves of these constituents after the ingestion of sugar and protein have been worked out and the limits of normality of these substances in the blood are well established. In a similar manner it is possible to determine the range of concentration of creatinin in the blood during health. The value of this information is considerable, since any departure from normal values will be an index to the type of disturbance of the metabolic processes.

The next step towards the complete elucidation of this difficult problem is the inquiry into the proportional concentration of the products of metabolism, more especially sugar, urea and creatinin. In this study consideration should be paid to the carbon dioxide content of the tissues and tissue fluids, since it is evident that oxidative changes must be influenced to a very great extent by the reaction of the medium and by the presence of buffering substances which control the reaction in spite of variations of the acid content. Many attempts have been made to gather some reliable facts in this connexion. Javal and others have sought for information by estimating the urea content of tissue fluids, such as cerebro-spinal fluid, the exudates in the pleura and in the peritoneum, and by comparing the results with the blood urea estimations. They came to the conclusion that the urea concentration of the body fluids and of the blood were practically identical. When the concentration was increased in the blood, an increase to a corresponding extent occurred in the tissue fluids. In a similar manner

Hagler and Schumann among others have determined that there is a close agreement between the sugar concentration of the blood and the sugar concentration of the tissue fluids.

Dr. Harold Rypins has recently investigated this question.¹ Dr. Rypins conducted a series of experiments to determine the concentration of metabolites during fasting, both in the blood and in the tissue fluids in persons suffering from pathological affections. He carried out careful investigations in connexion with thirty-six persons. When the concentration of the metabolites in the blood was within normal limits, the sugar concentration of pleural and peritoneal fluids ranged between 51.5% and 113% of that found in the blood. The urea concentration ranged between 37.5% and 180% and that of creatinin between 30% and 91%. One fluid, that of an empyema, has to be excluded, since it contained no sugar. Dr. Rypins yields to the temptation of expressing these figures as averages and endeavours to show that sugar is 83.42%, the urea nitrogen 80.08% and the creatinin 67.28% of the normal blood values. While his observations are of considerable value as individual measurements, it is not permissible to strike an average for twenty observations when the range is almost as wide as 400%. The figures lose in value since apparently only one reading was taken of each patient. It is quite possible that the sugar concentration of pleuritic fluid tends to remain at a level somewhat lower than that of the blood, but in order to demonstrate the fact, it would be necessary to have a number of readings of the same patient. It would then be manifest if variations of the blood sugar concentration were or were not followed by proportional variations of the sugar concentration in the exudate. In the case of the cerebro-spinal fluid the range of percentages determined by Dr. Rypins was also very wide. Dr. Rypins came to the conclusion that when the metabolite concentration of the blood was increased, the concentration in the tissues was increased in a proportionate manner. The same objection, however, holds good in regard to these figures as in regard to those previously discussed.

There are some significant points to which Dr. Rypins does not draw attention. It appears that six persons with pulmonary tuberculosis were included in the series. In two instances the peritoneal exudate and in four the pleural exudate were examined. The sugar content of the ascitic fluid was low, while that of the pleural fluid was high, higher than that of the blood. The urea content of both fluids and of the blood varied so greatly that no comparison could reasonably be made. The creatinin content was far less variable. It would appear that it is a mistake to include observations on patients suffering from many different diseases when dealing with so complex a subject. These figures will be valuable when they are supplemented by observations made by others and when these observations have been made continuously on the same patient.

¹ *Archives of Internal Medicine*, November 15, 1922.

Abstracts from Current Medical Literature.

DERMATOLOGY.

The Pathogenesis of Mercurial Stomatitis.

DANIEL BESSENE (Archives of Dermatology and Syphilology, March, 1923), after an extensive review of the literature on mercurial poisoning and the theories which relate to this, gives the result of his own experiments with regard to the causation of mercurial stomatitis. He maintains that of the nine theories put forward by different authors, not one can be regarded as sufficient to explain all the changes which take place, and that the local condition is only a part of a general intoxication. From the literature on the subject he draws the following conclusions: The mercury probably forms a compound with the blood, as indicated by an increased blood count and by hyperemia of the bone marrow. Reduction of the oxygen as a result of a decrease in the number of red cells and increase of lactic acid in the blood, together with the mercurial compound formed, act on the sympathetic system as an irritant and gradual asphyxiation results. Bacteria form hydrogen sulphide in the mouth and colon; this sulphide unites with the mercury in the blood and is deposited in the capillary endothelial vessels. This still further reduces the vitality of the tissues. Bacteria then act on the abraded surfaces of the mouth and colon and form ulcers at these points. The kidney, in an effort to throw off the mercury compounds, probably forms an albuminate which renders the cells less active. The idleness enforced by sickness and the inanition produced by poor absorption cause a loss of weight of the bones together with decalcification.

Leucoderma in Pityriasis Lichenoides Chronica.

HENRY MICHELSON (Archives of Dermatology and Syphilology, September, 1922) states that leucoderma may occur in the course of syphilis, leprosy, seborrhœic dermatitis and psoriasis; a few instances have been observed in pityriasis lichenoides chronica. The following is an instance of leucoderma occurring in pityriasis lichenoides chronica. The patient complained of a light brown eruption which commenced near the axilla and elbow; gradually the entire flexor surfaces became involved. On examination a diffuse, brown, lichenoid, papular eruption was seen to be present, somewhat resembling tinea versicolor. The patient's serum yielded no reaction to the Wassermann test. Eighteen months later the patient returned and said that the eruption had gradually become more intense. White areas had developed which were specially noticeable at the back of the neck and antecubital fossa. There were no subjective symptoms and there was no involvement of the mucous membranes. At this stage a positive reaction was

obtained from three Wassermann tests; subsequently no reaction could be obtained. Other instances of the same condition have shown a transient positive reaction in the serum to the Wassermann test. Treatment consisted in the use of ultra-violet rays and the injection of "Arsphenamin"; the latter seemed to have very little effect on the lesions.

Urticaria.

M. G. HANNAY (The Medical Press and Circular, January 31, 1923) discusses the causation of urticaria, which is regarded by many as an anaphylactic phenomenon. The chief points advanced in favour of this theory are as follows: The frequent occurrence of urticaria in cases of undoubted anaphylaxis; the fact that an urticarial subject or other members of the family will often be found to have suffered from asthma, hay fever et cetera; the analogy between urticaria and other recognized anaphylactic conditions; the minute quantity of offending substance often sufficient to induce an attack in a sensitized individual; the rapidity with which the symptoms may show themselves; the presence of eosinophilia as a feature in both anaphylaxis and urticaria; and the success of treatment by desensitization.

Treatment of Psoriasis.

H. E. ALSHWEDE AND W. BUSCH (The Urologic and Cutaneous Review, January, 1923) state that the most distinguished skin specialists have leaned for some considerable time towards the hypothesis that psoriasis is an infectious skin disease. Norwegian authorities have recently been emphasizing this view. Accordingly, efforts have been made of late to influence the course of the disease by raising the resisting powers of the body cells by means of a therapy consisting of parenteral irritation alone or with the use of germicidal agents. It has long been the practice to employ quinine in psoriasis on account of the results obtained in malaria. The hitherto unsatisfactory results are claimed by the authors to be due to using too much quinine over a prolonged period.

Tissue Reaction in Malignant Epithelioma of the Skin.

HOWARD PARKHURST (Archives of Dermatology and Syphilology, October, 1922) calls attention to the value both in prognosis and diagnosis of the tissue reaction in malignant epitheliomata of the skin. For the purpose of investigation the author has examined sections from over one hundred instances of epitheliomata in human beings and noted the type, whether basal or prickle-celled, its age and rate of growth and the depth to which the underlying corium was invaded. He also noted the special characteristics of the stroma, of the nuclei and the amount of connective tissue degeneration, together with the number of plasma cells and lymphocytes

varies directly with the amount of hyaline degeneration. He also concludes that, as the number of plasma cells is an indication of the degree of resistance of the tissues, the malignancy of the neoplasm varies in inverse proportion to the number of plasma cells. A third conclusion is that the more malignant the process, the less the number of plasma cells.

Chronic Eczema and Concentrated Carbon Arc Light.

SVEND LOMHOLT (The British Journal of Dermatology and Syphilis, February, 1923) reports that, owing to the variable results and the risk incurred in the treatment of chronic eczema with X-rays, a series of experiments have been carried out at the Finsen Institute on the use of concentrated carbon arc light in place of X-rays, with the result that 60% of the patients were permanently cured. The percentage of those who were more or less improved, was twenty-five; the remaining 15% showed little if any change. No instance occurred in which the condition was aggravated. The old infiltrated and keratotic types of eczema, those generally characterized by the name of chronic neuro-dermatitis or lichen chronicus, are most suitable for light treatment. In addition, many vesicular forms were influenced by this treatment.

Calcification of the Skin.

R. R. DUCASSE (Archives of Dermatology and Syphilology, March, 1923) divides calcification of the skin into two main classes, calcification associated with neoplasms and a generalized type associated with other pathologic processes in the skin. The instance of calcification reported by the author appears to have arisen without any previous skin lesion and is therefore atypical. The eruption consisted of lesions varying in size from that of a pinhead to a pea on the extensor surfaces of the upper and lower extremities. These frequently coalesced and formed tumours of about three centimetres in diameter. At varying periods inflammatory reactions supervened, accompanied by a softening of the mass and an extrusion of white paste through the sinuses which on closing were followed by scarring. Treatment apart from radium has been highly unsatisfactory.

Lupus Erythematosus.

P. E. BECHET (Archives of Dermatology and Syphilology, September, 1922) reports a typical instance of lupus erythematosus. The prominent, patulous follicles, scales and atrophy were all present. The condition had been present for over a year from October, 1921, to January, 1923. The surface of the lesion was painted with 95% phenol solution which was thoroughly rubbed in with a cotton swab. Five or six applications were made in all, at intervals of two weeks. At the time of reporting the case the nose was thoroughly healed and there was no evidence of scarring.

RADIOLOGY.

Spark Gap Measurements.

K. EDGUMBE (*Journal of the Roentgen Society*, October, 1922) describes the construction of the various instruments used in the measurement of electrical discharge. After referring to various instruments of the moving coil type, he refers to the method of measurement of high tension current by means of the spark gap. The spark gap in its most accurate form consists of two spheres of large diameter, capable of being fixed at various known distances apart. As long as the diameter of the spheres is at least 50% greater than the gap which separates them and as long as they are kept well away from other objects (at least three times the gap length), the sparking voltage is extremely constant and is usually considered to depend upon the peak value. With spheres whose diameter is twenty-five centimetres, the sparking distances are thirty-five millimetres for one hundred kilo-volts, fifty-six millimetres for one hundred and fifty kilo-volts, seventy-eight millimetres for two hundred kilo-volts and one hundred and forty-one millimetres for three hundred kilo-volts. The point gap is often used to measure voltage, but is subject to many errors and cannot, in any case, be regarded as a reliable apparatus for voltages exceeding thirty to forty kilo-volts. The chief objection to the point gap is that, owing to the sharpness of the points, a glow discharge, called a "corona," forms at voltages much below that of breakdown. This discharge ionizes the air and thus tends to irregularities in the breakdown voltage. The gap is very sensitive to disturbance by neighbouring objects and, for this reason, all other bodies, including the supports, must be kept a considerable distance from it. Again, the length of the gap for a given voltage is about four times as great with needle points as with spheres. A two hundred kilo-volt spark gap, for example, would occupy a space of about 2.4 by 1.2 metres (eight by four feet) and if this is compared with the spark gaps commonly employed, the reason for the notorious unreliability of the indication of the latter will be evident. It is always advisable to connect a resistance in series with the gap, whether sphere or point, so as to limit the current which flows at the moment of breakdown to one-tenth of an ampere or less. A resistance of a megohm at one hundred kilo-volts is therefore suitable and serves two purposes. It protects the plant from damage and the spheres from the pitting which occurs in the event of a heavy discharge current; but, above all, it prevents the occurrence of surges at the moment of sparking over, which are so liable to damage other apparatus connected to the circuit. A carbon rod resistance is suitable for the purpose.

Tonsil Radio-Therapy.

C. A. WATERS, P. B. MACREADY and C. H. HITCHCOCK (*American Journal of Roentgenology*, August, 1922) deal with the problem of X-ray treatment

of chronically infected tonsils and adenoids. They endeavoured to solve the problem of whether the same clinical results could be accomplished as in removal and whether there was a persistence of the "carrier" state. Swabs were taken before and during treatment and the distance between the two tonsils was also recorded. A series of four exposures were given (twice weekly for two weeks) and after an interval of several weeks the dosage was repeated. Dosage consisted of five milliamperes of current filtered through four and a half millimetres of glass and one and three-quarter centimetres of orthopaedic felt, at 120,000 volts and twenty-five centimetres focal distance for a time period of five minutes. After the first four exposures a period of four weeks elapsed before repetition of dosage. This dosage caused a mild erythema, which rapidly disappeared. The tonsils were greatly reduced in every instance, but the bacteria were unaltered and the "carrier" state persisted. The authors consider that tonsillectomy is preferable to radio-therapy as the infected foci, together with the flora, are extirpated. When tonsillectomy is contra-indicated, radio-therapy is of great value, as in chorea and in cardiac and renal lesions.

Cancer Grafts and Radiation.

H. LIU, E. STURM and J. B. MURPHY (*Journal of Experimental Medicine*, April 1, 1922) contribute an original article on the fate of cancer grafts implanted in subcutaneous tissues which have been previously subjected to radiation. The authors refer to the frequent advocacy of radiation to the open wound after breast removal, but it is difficult to appraise its value, as it is impossible to make any efficient control exposures. Several authors have shown that cancer cells *in vitro* are not killed by much greater doses than the human skin can stand, but that a much smaller dosage than this lethal dose will greatly increase the resistance of the skin and subcutaneous tissues to cancer implantation. In the authors' experiments, a dosage of ten milliamperes at a 7.5 centimetre gap for two and a half minutes at a distance of fifteen centimetres was used. Large flaps were turned back and the area radiated and then the transplants were made. It was found that such irradiated areas were very resistant to cancer implantation. Numerous interesting experiments were undertaken to show that the erythema dosage did not extend deeply into the underlying tissues. Excessive dosage to the animals was found to lower ordinary resistance and the power of cancer resistance.

Cancer of the Cervix Uteri.

LEON GERARD (*La Consultation*, March, 1922) describes the value of radium in the treatment of cancer of the cervix uteri. For successful treatment it is necessary to make early diagnosis and this is often a matter of extreme difficulty. The disease may be divided into three stages. The first stage shows a simple erosion with an indurated base, the second stage shows

either a fungating surface or a deep erosion involving the neck and possibly part of the vaginal vault and the third stage is an advanced one where the condition involves the rectum and bladder and parametrium. Diagnosis should be made by microscopic section. Where the disease is localized to the uterus, the condition should be treated by radium, but when the parametrium is involved it is necessary to combine surgery and radiation. Screened radium tubes are placed in the cervical canal, one in the anterior and one in the posterior fornix and one on each side of the cervix. The tubes are held in place with a gauze tampon. The dose given is sixteen millicurie hours. Cachexia and gland involvement are contra-indications to radiation.

Gall Bladder Radiography.

B. R. KIRKIN (*The American Journal of Roentgenology*, November, 1922) writes on the diagnosis of gall bladder lesions by X-ray examination. The normal gall bladder cannot be demonstrated on a skiagram. A gall bladder, therefore, that is demonstrable must be pathological. Further, a single gall stone may cast no shadow, but when a collection is present the combined shadows may be visible. The author advises the administration of an opaque meal, as several indirect signs may point to the probability of a gall bladder lesion. The gall bladder may cause a definite pressure defect on the stomach or duodenal shadows and the hepatic flexure may be displaced by gall bladder pressure. Adhesions around the gall bladder may also cause deformities of the gastroduodenal or colic shadows. Gastric hypermotility is common in gall bladder disease. Several skiagrams are taken with rays of varying penetration and all plates are developed for the same period of time. In this way skiagrams of varying density are obtained and frequently a gall bladder shadow will be visible in one and not in another. The author has examined seven hundred and twelve patients and the skiagraphic findings of gall bladder pathology were confirmed in 93.15% of the series.

Fibrocystic Osteitis.

C. P. G. WAKELY (*Archives of Radiology and Electrotherapy*, January, 1923) reports an instance of fibrocystic disease of the upper end of the humerus. The author comments on the increased knowledge of this disease in late years, although Virchow described an instance in 1877. The patient described by the author was sixteen years of age and complained of pain in the shoulder. There was no history of injury. The upper end of the humerus was palpably enlarged. A skiagram showed a definite cystic condition near the upper epiphyseal line. The cyst was scraped out and showed no cartilage or giant cells. A small piece of bone which was removed, showed absorption by osteoclasts and a thin layer of osteoid tissue. Seven years later the patient was quite well and a skiagram showed complete regeneration of the humerus.

Special Abstract.

THE REACTION OF THE BLOOD.

THE HÆMOGLOBIN COMMITTEE OF THE MEDICAL RESEARCH COUNCIL, comprising Dr. J. Barcroft, F.R.S., Professor Sir William Bayliss, F.R.S., Dr. C. G. Douglas, F.R.S., Dr. H. H. Dale, F.R.S., Professor G. A. Lovatt Evans, Professor F. R. Fraser, Mr. W. B. Hardy, F.R.S., Dr. H. Hart-ridge, Professor Laurence J. Henderson, Professor A. V. Hill, F.R.S., and Dr. Poulton, has undertaken an intensely difficult task. It has attacked the problem of the acid-base equilibrium of the blood and has set out in the course of a report covering some sixty-nine pages the facts that have been disclosed concerning this highly complex subject.¹ The Committee has performed a very useful service in reducing the doctrines of the several workers to terms conveying one definite meaning each. The report thus becomes a reliable record of the present position of knowledge.

The Hydrogen Ion Concentration.

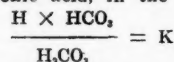
In order to define the acid-base equilibrium of the blood, it becomes necessary to apply every measurement that has been devised for this purpose, since the complicated physico-chemical condition of the fluid presents certain obstacles to the uncontrolled acceptance of results based exclusively on one method of expression. According to the electrolytic dissociation theory pure water is dissociated into two equally but oppositely charged ions, H and OH. The concentration of hydrogen ions set free is 10^{-7} grammes per litre at 22° C. This dissociation increases with a rise of temperature. Similarly the equivalent of hydroxyl ion per litre may be expressed as 10^{-7} grammes. In other words, pure water at 22° C. may be regarded as a ten millionth normal acid and a ten millionth normal alkali. When acid or alkali be added to water the product of the concentration of the two ions is unaltered, since while the hydrogen ion in the one case or the hydroxyl ion in the other is increased, the other ion is proportionately reduced by a depression of the ionization reaction of the water. The product of the two ions is necessarily 10^{-14} . If strong acid is added, so that the solution is a one in a hundred thousand normal acid, the hydrogen ion concentration would be 10^{-4} . But the hydroxyl ion concentration would be correspondingly reduced to 10^{-10} , thus bringing the product again to 10^{-14} . The same obtains in any solution of acid or alkali. It follows that the clumsy expedient of employing negative symbols can be avoided, which obtain if the acid-base equivalent be expressed either as concentration of ions or as normality of ion, if some conventional form be substituted. Sørensen suggested that, instead of expressing the concentration of hydrogen ions, cH of a 1:100,000 normal acid solution as 10^{-4} , the formula $\text{pH} = 5.0$ be employed. The conversion is effected by the aid of logarithms. In the next place, it is found that at 22° C., when the pH value is less than 7 the solution is acid; when it is 7, there is a neutral reaction and when it is greater than 7, the solution is alkaline. At body temperature neutrality is expressed either as $\text{cH} = 1.83 \times 10^{-7}$ or $\text{pH} = 6.74$.

Buffer Substances.

If a solution of an acid be neutralized by the gradual addition of solid hydroxide of an alkali, the hydrogen ion concentration of the solution would diminish nearly in proportion to the amount of base added until the indicator would show a sudden change. If urine or blood serum be neutralized with the help of an indicator, the change appears sluggish and the end point is uncertain. If methyl red and phenolphthalein be used side by side, it will be seen that the former will indicate a lower degree of acidity than the latter. By direct measurements it has been found that the change of hydrogen ion concentration follows a curve and is not a straight line like that of a simple neutralization of acid by alkali. Moreover, it has been ascertained that simple dilution of serum or urine

does not lead to a material alteration of the hydrogen ion concentration. Similar results are noted when a solution of glycine or of a phosphate is titrated. This anomalous behaviour is attributed to the presence of "buffer" substances. If a small quantity of a strong acid be added to a solution of sodium acetate, nearly all of it will be replaced by the equivalent quantity of acetic acid. Acetic acid is dissociated to a relatively small extent. Moreover, as long as any sodium acetate remains, the acetate ions will depress the ionization of the weak acetic acid. For these two reasons the increase of hydrogen ions produced by a given addition of a strong acid to an acetate solution is much less than that produced by the same addition of water. The salts of weak acids are the most effective buffers against the addition of acid, while the salts of weak bases are the most effective against alkalies. Further, it has been found that these salts are hydrolytically dissociated in solution in water. Sodium chloride, a salt of a strong acid and a strong base, is split almost entirely into sodium and chlorine ions and the reaction remains neutral. Sodium acetate in solution contains free acid and base in addition to the ions. The base is strongly dissociated, but the acid is not and in consequence the solution has an alkaline reaction. Theoretically a fully buffered solution would suffer no change in the concentration of its hydrogen ions by the addition of acid or base. Although such a condition does not exist, partial buffering occurs under many circumstances.

In blood plasma carbonic acid acts as the acid and sodium bicarbonate as the base. The concentration of hydrogen ions of plasma depends on the relative concentration of dissolved carbonic acid and of bicarbonate. The ratio is roughly 1:20. The sodium bicarbonate is approximately in a 0.03 molar solution and the carbonic acid in a 0.0015 molar solution. Sodium bicarbonate in 0.03 molar solution and in equilibrium, with a tension of carbon dioxide sufficient to prevent appreciable dissociation of carbonate, has a pH of about 10. But the pH of plasma is less than 6.74. The tension of the carbonic acid in plasma is never as low as this level, nor does it reach the level of atmospheric pressure. The law of mass action is held by Henderson to express the dissociation of a weak acid, such as carbonic acid, in the following equation:



In this equation K represents the dissociation constant or the ratio of the product of the concentration of the ions to the concentration of the non-dissociated acid. Henderson showed that when bicarbonate of soda is present as well, only a small part of the anions is derived from the carbonic acid, the greater part arising from the electrolytic dissociation of the bicarbonate. He has therefore set up a further equation in which δ represents this electrolytic dissociation. By combining the two constants, K and δ , the result may be expressed in one term, K_1 :

$$\text{H} = \frac{\text{K}(\text{CO}_2)}{\delta(\text{NaHCO}_3)} = \text{K}_1 \frac{(\text{CO}_2)}{(\text{NaHCO}_3)}$$

The Committee call attention to the fact that in blood the sodium bicarbonate represents the total concentration of carbonic acid either combined with or neutralized by sodium or potassium and that this is not necessarily independent of the carbonic acid pressure, since the base may be derived from combinations with other weak acids by increasing the carbonic acid pressure. They give a series of equations by means of which the pH or pK₁ can be expressed as — log. H. or — log. K₁. Some ingenious work has been carried out by Hasselbalch, but the results of the earlier work were found to be inexact. In these calculations the volume of the fixed carbonic acid per hundred volumes is taken into account. This is expressed by the symbol B, while the solubility of the carbonic acid gas at the given temperature is expressed as α . The final formula expressed in pH terms is as follows:

$$\text{pH} = \text{pK}_1 - \log. \frac{3.8 \text{ B}}{\alpha}$$

It would appear that according to this formula the value of pK₁ should be approximately 6.1. To calculate from this the pH value, it is necessary to ascertain the ratio

¹ "The Acid-Base Equilibrium of the Blood," Special Report Series, No. 72; 1923. Medical Research Council of the Privy Council. Price: 2s. net.

between the free and the fixed carbonic acid. This acid is the only acid of importance in the blood, for there is sufficient bicarbonate to combine with other acids which are stronger. It is true that the acid radicles of protein are weaker acids and they may exercise a varying influence on the dissociation.

The authors of the report find that some confusion exists in regard to the conception of the term reaction of the blood. If true plasma be taken, that is, plasma separated from corpuscles, but under the same given partial pressure of carbonic acid as it was when it was in contact with the corpuscles, it is found that the reaction is unaltered. At lower carbonic acid pressures whole blood is less alkaline than separated plasma, while at higher pressures the reverse is true. This means that whole blood is a better buffer than plasma.

Ketosis.

Formerly the term acidosis was employed to indicate the condition associated with the presence of certain acids in the urine in diabetes. These substances are β -oxybutyric acid, aceto-acetic acid and acetone. Acetone is probably produced by the separation of carbonic acid from aceto-acetic acid. The latter body as well as β -oxybutyric acid is supposed to be produced from the secondary alcohol and ketone groups by oxidation and reduction. The Committee hold that it will be advisable to discard the term acidosis and to employ the term ketosis for this condition. When the blood is less alkaline than normal, they use the term acidemia and when it is more alkaline they use the term alkalemia. These terms are employed solely in reference to the reaction of the blood; the presence or absence of ketosis is immaterial.

Van Slyke introduced the term alkali reserve, which signifies the volume of carbonic acid that can be expelled by acid from one hundred volumes of blood or blood plasma which has been brought into equilibrium with carbonic acid at forty millimetres pressure. The carbonic acid held in simple solution must be deducted for this calculation. It is obvious that this is in fact a measurement of the bicarbonate content of the plasma. It has been suggested that carbon dioxide combines directly with hæmoglobin. Others, however, have challenged this, since they have shown that hæmoglobin acts as an acid and not as a base. It has further been shown that the bicarbonate content of the blood may be altered by the movement of alkali between the blood and the tissues. For these and other reasons it is held by the Committee that the term alkali reserve is misleading. They prefer to call the measure of the volume of carbon dioxide displaced by acid from the blood the carbon dioxide fixing power or the fixed carbon dioxide.

They give information concerning the work carried out in connexion with the titratable alkalinity of the plasma.

Variables in the Acid-Base Equilibrium of Plasma.

Various workers have devised curves to depict certain qualities of the blood in relation to its acid-base equilibrium. These include the oxygen dissociation curve, the combined carbonic acid dissociation curve, the carbon dioxide curve, the buffer curve, the carbon dioxide reaction curve, the so-called Henderson-Adair line and the pH — log. K relation curve.

In concluding the first part of the report the Committee reproduce Joffe and Poulton's diagram of the carbon dioxide dissociation curve of human blood, a compound graph that has been found of much value in determining the hydrogen ion concentration of blood and blood plasma under various conditions of carbon dioxide tension and fixed bicarbonate content.

Measurement of the Reaction of Blood.

The Committee preface a special section dealing with the determination of the reaction of the blood with a short chapter on the taking of blood samples. Arterial blood differs from venous in that it contains less bicarbonate, less carbon dioxide, more water and a somewhat smaller volume of corpuscles, while its hydrogen ion concentration is lower. While much is known concerning the physico-chemical causes of these differences, it is not yet possible to calculate with accuracy the composition of arterial blood from an examination of venous blood. It is consequently of advantage to collect samples of

arterial blood for the purpose. It will be recognized that arterial blood is less variable in composition than venous blood. Hürthler showed that with care a sample of blood can be withdrawn safely from the radial artery. The sample is collected in potassium oxalate to prevent coagulation. This does not disturb the carbon dioxide content or capacity or the oxygen content or capacity or the reaction of the blood.

The measurement of the ionic concentration of a solution is best carried out by measuring the electro-motive force between the solution and the metal electrode and translating the result by means of a formula. When it is required to measure the hydrogen ions, the hydrogen electrode is employed. The principle on which this electrode is based, is that hydrogen gas is absorbed by finely divided platinum or palladium to form an electrode which behaves just like a metallic amalgam electrode. The electro-motive force between the solution and the electrode is computed by putting the hydrogen element in series with a standard electrode of known electro-motive force. In this way a complete galvanic cell is constructed of which the electro-motive force can be measured in the usual way with a potentiometer. But there are many peculiar difficulties to be overcome when this method is applied to blood. In the first place, a known pressure of hydrogen is required in the fluid and above it. The blood must therefore be deprived of its carbon dioxide and oxygen and this necessarily alters the reaction. A method has been devised by which a hydrogen-carbon dioxide atmosphere is used in the place of the pure hydrogen atmosphere. Other difficulties can be overcome, but the processes are tedious to perform and it is not certain that the measurements are more reliable than those of other methods.

The next method described is the indicator method. Bayliss introduced a simple device for approximate determination. The blood is centrifuged and a known quantity of neutral red is added. The colour is matched to a standard solution of phosphate containing neutral red. At times allowance must be made for the presence of pigment and hæmoglobin. Several other, more exact methods are described. In the indirect methods use is made of the fact that the reaction of the blood is dependent on the ratio between fixed to free carbon dioxide. It has to be remembered that if the bicarbonate content of the blood is reduced, the blood will become more acid. As a result of this change a lowering of the carbon dioxide will bring about a restoration of the reaction to nearly the original level. The compensation is not complete, because as soon as the breathing returns to normal, the carbon dioxide pressure must rise and the decreased alkalinity must again appear. Hasselbalch's method is briefly as follows: Red blood corpuscles are washed and dialysed for three hours. They are then made up with sodium bicarbonate to make a solution of 0.025 normal sodium bicarbonate at between 7 and 94.2 millimetres pressure of carbon dioxide. It is found that this solution contains less carbon dioxide than a 0.025 normal sodium bicarbonate solution not containing hæmoglobin. By placing blood in a saturator in a water bath at 38° C., any given pressure of carbon dioxide can be obtained. The blood can be removed from the saturator without loss of carbon dioxide and the volume of the carbon dioxide in the blood can be calculated by one or several recognized methods. Values for pK_1 are given for varying carbon dioxide pressures. From these the pH can be calculated. A full description is given of the application of Hasselbalch's method. In the next place the Committee deals with Van Slyke's method, which consists in separating the plasma from oxalated blood after centrifugalizing, bringing the plasma into equilibrium with the alveolar air of the observer of room temperature and estimating the carbon dioxide content by means of Van Slyke's apparatus. This method reveals the bicarbonate content of the plasma at a standard pressure of carbon dioxide of forty millimetres. The authors of the report find that the determinations are only of value if the Hasselbalch formula in one form or another is applicable to the particular blood specimen under consideration.

In carrying out these measurements it is of fundamental importance to have a method of estimating with a high degree of accuracy the concentration of free carbon dioxide in the blood. It has been shown that as a result of the diffusion of gases through the pulmonary epithelium, the carbon dioxide in the alveolar air is practically in equilibrium

brum with the carbon dioxide of the blood. In these circumstances the alveolar carbon dioxide pressure has been used for the purpose of determining the free carbon dioxide in the blood. While it would appear that under normal conditions this measurement is reliable, there is a remarkable discrepancy between the arterial and the alveolar carbon dioxide in many pathological conditions. Two explanations are offered. In the first place, in some pathological conditions the patient is incapable of a sufficiently deep expiratory effort to insure a true sample of alveolar air. In the next place, shed blood, especially if kept at 37° C., suffers a progressive loss of the combining power of its carbon dioxide. It thus appears that caution should be exercised before the alveolar air is accepted as an index of the free carbon dioxide of the blood, save in normal persons.

Barcroft found that the oxygen dissociation curve of blood depends on the hydrogen ion concentration of its plasma, while the dissociation curve of a hæmoglobin solution depends on the hydrogen ion concentration of the solution. By the addition of acid the curve is shifted, so that less oxygen is taken up at any given oxygen pressure. In the case of a hæmoglobin solution the same obtains. It is the same whether the alteration in the hydrogen ion concentration is brought about by carbonic acid or by a fixed acid like hydrochloric acid. By utilizing Hill's formula for an oxygen dissociated curve,

$$\frac{y}{100} = \frac{Kx^n}{1 + Kx^n}$$

in which x is the oxygen pressure, y is the percentage saturation and n is a constant (in the case of blood usually about 2.5), Barcroft and Peters found in given investigations that the relation of pH to log. K was a straight line. This may be expressed in another way. The value $1/K$ is proportional to some power of cH . Now oxyhæmoglobin is a more highly dissociated acid than reduced hæmoglobin. Hill showed that the relation $1/K = acH$ should be approximately true over a certain range of cH , if pH be regarded as the hydrogen ion concentration within the corpuscles. This holds good on the assumption of an identity or proportional value of the hydrogen ion concentration of the plasma and that of the corpuscles. Some small divergencies noted are probably explicable, since the n values are often too low. It appears, however, that the constant a is not the same for different samples of blood, when cH is regarded as the hydrogen ion concentration of the plasma. Barcroft has put forward the view that the hydrogen ion concentration of the corpuscular contents is not the same as that of the plasma. Barcroft would adopt the value $1/K$ as the cH of the corpuscular contents. This matter is discussed at some length. The conclusion is reached that, while the hydrogen ion concentration of the plasma is not the same as that of the corpuscles, it is proportional to it over the range of the hydrogen ion concentration of physiological importance. It is permissible to use the value K to measure variations in the hydrogen ion concentration of plasma in such conditions as exercise or oxygen want, even if this is not so for the purpose of ascertaining absolute values under physiological conditions.

The Buffers of the Blood and Tissues.

The authors of the report recognize that the study of the reaction of the blood involves two separate problems, the mechanism of the buffering of the shed blood and the mechanism of the neutrality regulation in the living body. They deal with the first problem in an exhaustive manner before turning to the second. In attacking the problem of physico-chemical properties of the blood from the point of view of the buffer substances, they take into consideration the phosphates, the bicarbonates and the proteins. Di-sodium hydrogen phosphate is regarded as the sodium salt of the feebly acid sodium di-hydrogen phosphate. Addition of acid causes the relative concentration of the latter to be increased at the expense of the former. But since the actual amount of inorganic phosphate in the plasma is extremely small, it is obvious that it plays but a negligible part in the buffering of plasma.

The position of the bicarbonates, however, is quite different. Sodium bicarbonate, being the sodium salt of the weak acid, carbonic acid, is an effective buffer, because carbonic acid is set free by the action of a stronger acid

which is dissociated to a small degree. The carbon dioxide gas escapes from solution when exposed to a tension of this gas lower than the tension in the tissues. This liberated carbon dioxide is removed by pulmonary ventilation. Arterial blood plasma has a content of fixed carbon dioxide corresponding to a bicarbonate concentration of about 0.03 molar solution. The sodium bicarbonate can be removed from plasma by dialysis. This substance is at times spoken of as the available base, because it represents the amount of base available to neutralize an added acid. It is found that the reaction of plasma is not that of an aqueous solution of sodium bicarbonate of identical carbonic acid and sodium bicarbonate content, since the sodium chloride of the plasma depresses the degree of the ionization of the bicarbonate and thus leads to an increase of hydrogen ion concentration. By actual measurement the pH of plasma is found to be 0.3 lower than that of a corresponding solution of carbonic acid and sodium bicarbonate without other salts.

There is about 8% more protein in plasma than bicarbonate. The molar concentration of the proteins, however, is considerably smaller than that of the bicarbonates. Proteins, in virtue of their basic and acidic groups, are capable of combining with both acids and bases. The protein molecule possesses seven or eight groups potentially yielding free ammonia. The molar concentration of these basic groups in the proteins of plasma has been estimated as about 0.05. It thus appears that plasma protein has buffering properties capable of competing with bicarbonate. Further study, however, has brought some highly interesting limitations of this buffering quality of proteins into view. It has been found by experiment that at a particular hydrogen ion concentration no migration of the protein in the electric field can be observed. The anions and cations are in equal concentration. This is the so-called isoelectric point. Usually the point is somewhat on the acid side of neutrality. The conditions obtaining in a protein solution containing sodium chloride or other ions are very complicated. When a curve is plotted of the change in a protein solution in its relations to its hydrogen ion concentration, it is noted that the changes are scarcely perceptible for some distance on either side of the isoelectric point. Various explanations have been offered for this behaviour, but the inevitable conclusion that all observers have adopted, is that the proteins have extremely little action as buffers in plasma.

Hasselbalch has produced evidence to show that whole blood is more extensively buffered than plasma. The additional buffering in secondary buffering is due to the presence of the corpuscles. When blood is exposed to an increasing concentration of carbon dioxide, the bicarbonate content of the plasma is increased and the chloride content is reduced. Joffe and Poulton have set out in tabular form the fixed carbon dioxide values of true plasma and of separated plasma at varying pressures of carbon dioxide. From this table it is seen that, while the bicarbonate content of separated plasma at low carbon dioxide pressures (between ten and twenty millimetres) is much greater than that of true plasma at the same carbon dioxide pressures, the values are approximately the same at forty millimetres. At higher pressures the bicarbonate content of true plasma exceeds that of separated plasma. It has recently been shown that there is no extensive displacement of basic ions from the corpuscles to the plasma as the carbon dioxide pressure increases. The view at present commanding adherence is that, when blood is exposed to low carbon dioxide pressures, the available base of the corpuscles is largely united to the hæmoglobin. At the same time the bicarbonate concentration of the plasma is low. If the carbon dioxide pressure be raised, minute traces of hydrochloric acid appear as a result of the reaction between the sodium chloride of the plasma and the carbonic acid. The trace of strong acid is taken up by the corpuscles and this in turn leads to a disturbance of the equilibrium of the plasma, with the formation of more hydrochloric acid. This process continues until the corpuscles are unable to absorb any more acid. Equilibrium is reached at this stage between the pH of the plasma and that of the corpuscles. The authors amplify this argument and refer to the most important work done on the question.

In the next place they establish the axiom that oxyhæmoglobin possesses a greater power of fixing carbon dioxide

than does reduced hæmoglobin. This means that the former is a stronger acid than the latter. Great importance is attached to the fact that carbon dioxide and oxygen tend to displace one another from the blood. Henderson and Adair have evolved a method of expression of the effect of carbon dioxide in altering the oxygen dissociation curve of the blood in a graphic manner and have shown that there is a linear relation between $1/K$ and the carbon dioxide partial pressure. In other words, the reaction of the contents of the corpuscles can be shown to run parallel to the hydrogen ion concentration of the plasma. At the low oxygen and carbon dioxide partial pressures obtaining within physiological limits, the transit of oxygen to the tissues is facilitated when the carbon dioxide pressure is increased. It thus appears that in the change from the arterial to the venous condition the alkalinity of the blood is reduced to an extent greater than can be explained merely as a result of an increase of carbon dioxide.

The Nomogram.

L. J. Henderson has undertaken the immense task of illustrating the complex chemico-physical conditions of the blood in one compound graph. There are six important variables that must be taken into consideration; perhaps there are many others. The first is the free oxygen of the blood and the second the combined oxygen of the blood. The third is the free and the fourth the combined carbon dioxide of the plasma. The fifth is the hydrogen ion concentration of the plasma and the sixth the chloride concentration of the plasma. These variables are plotted in Henderson's nomogram, a graph with multiple ordinates and abscissæ. At first sight it is extremely difficult to follow the bi-dimensional indications and to recognize the full significance of values at a given set of variables. The authors assist the reader materially by explaining the readings at certain levels. It would be impossible to convey an intelligible impression of this graph and its working in a condensed abstract. Readers interested in this particular method of expression must therefore either study Henderson's original publications or the admirable summary given by the authors of the report.

The Buffering of the Tissues.

It will be realized that as carbonic acid is the end product of metabolism, the reaction of the tissues could be kept constant by the removal of this product. Actually, however, there are many intermediate products that remain in the tissues under normal conditions. These products may find their way into the blood as a result of excessive work or of some modification of normal metabolism. The ordinary tissues produce acid out of neutral precursors. In these circumstances it seems that the tissues need to be buffered, as part of the general scheme of equilibrium. Unlike blood, the tissues contain considerable quantities of phosphates, while red muscle contains hæmoglobin. It is probable that the relation between muscle fibres and the surrounding tissue fluid is similar or analogous to the relation of corpuscles and plasma. There is reason to expect that the proteins and the phosphates take an active part in the maintenance of the neutralization of lactic acid formed in contracting muscle.

The Estimation of the Hydrogen Ion Concentration of Normal Blood.

In the second part of the report, the authors deal to some extent with the relation of the changes in the hydrogen ion concentration of the blood to certain pathological conditions of the body. In approaching this difficult and still obscure subject, they find it necessary to adopt some expression of the hydrogen ion concentration of blood said to be normal. From what has been given below, it will be gathered that all measurements have to be scrutinized with critical care before they can be accepted as reliable. Fraser, Ross and Dreyer have employed dialysis and comparison against indicators to blood collected by arterial puncture. The extreme limits of pH found were 7.72 to 7.64, that is, a range of 0.08. Barcroft, Bock, Hill, Parsons and Shoji have used the hydrogen electrode. They collected venous blood and reduced it. The blood was equilibrated against forty millimetres of carbon dioxide. The extreme limits of pH were found to be 7.47 to 7.34. Sonne and Jarlöv measured the ratio of free to combined carbon dioxide in blood equilibrated at forty millimetres.

The extreme limits disclosed by this method were from 7.33 to 7.28. The authors exercise caution in regard to the reliability of these readings. They recommend workers who wish to estimate the pH of blood to select one method, to attain familiarity with the apparatus and the solutions and to practise the determinations on samples of blood from normal people. In this way they will probably obtain results reliable for differences of 0.05 pH or more. It has been shown by several competent observers that after the performance of work the pH values may become lowered by 0.05, 0.1 or even 0.15. Hasselbalch could find no detectable variation of the reaction of the blood at alveolar pressure during pregnancy, although the carbon dioxide fixing power was reduced. In certain pathological conditions some remarkable deviations from the normal have been recorded. Dale and Evans, working with animals, have measured pH = 9.0 on the one hand and pH = 7.22 on the other. It is true that the animals either died within a short time of the experiment or were killed, but the fact that the readings were obtained during the life of the animals is important.

The authors endeavour to approach the question of the significance of an altered reaction of the blood. They adopt the device that has appealed to almost every biophysicist who has been engaged in this study. They start with the symptom of dyspnoea and inquire whether it is associated with an increased hydrogen ion concentration of the blood. They recognize, however, that if this be the case, it does not necessarily follow that the symptom is caused by this change.

It has long been known that hyperpnoea is caused by the conditions that lead to an increase of venosity of the blood passing through the respiratory centre. Haldane and Lorraine showed that the respiratory centre is far more sensitive to increase of carbon dioxide than to oxygen deficiency. During natural breathing at rest the alveolar carbon dioxide pressure remains constant in spite of any ordinary increase in barometric pressure. Haldane further found that if air containing a small proportion of carbon dioxide were breathed, an increase in the carbon dioxide pressure in the alveoli maintained at a level just above the constant normal level for that individual would suffice to double the resting breathing. He further found that a maintained decrease of the alveolar carbon dioxide pressure below normal by two millimetres reduced the activity of the respiratory centre to zero. This means that it produces apnoea. Boycott and Haldane showed that any acid was capable of stimulating the respiratory centre, while Winterstein produced evidence which indicated that the degree of stimulation was proportional to the hydrogen ion concentration. Some very interesting experiments were conducted by Hasselbalch on the variations in the hydrogen ion concentration of the urine under the influence of alterations of diet. He found that when a person existed on a vegetable diet, the resting alveolar carbon dioxide pressure was a few millimetres higher than when the individual was taking a meat diet. The vegetable diet tended to reduce the hydrogen ion concentration of the blood and as a result the activity of the respiratory centre was slightly diminished. This led to an increase in the carbon dioxide pressure, both in the alveolar air and in the arterial blood. The carbonic acid-bicarbonate ratio became nearly restored. Conversely he showed that when a person was living on a protein diet, there was a change in the reverse direction. The respiratory centre apparently responds to minute changes in the hydrogen ion concentration of the blood reaching it.

The problem of the effect of the secretion of acid or alkaline digestive juices is also considered. In this, as well as in many other conditions involving alterations in the hydrogen ion concentration, the compensatory mechanism is brought into play. These changes have been demonstrated after the ingestion of bicarbonate and of ammonium chloride. It thus becomes clear that a process described as physiological buffering must be recognized in addition to the physico-chemical buffering. Finally, it should be noted that the behaviour of the cell membrane and of protoplasm of the cell also influences the adjustment of the reaction. Jacobs has shown that the internal reaction of cells can be rendered acid much more effectively by free carbon dioxide than by other acids. He holds that the cell membrane is easily permeable to un-

dissociated CO_2 , (H_2CO_3) and relatively impermeable to hydrogen ions. Since the reaction of the blood is determined by the ratio of free to combined carbon dioxide, any change of reaction, even if caused by the addition of fixed acid or alkali, is traceable to a change of this ratio. The authors state that if attention is directed to the changes of cH in the nerve cell of the respiratory centre, it is not impossible that evidence as to the relative importance of free carbon dioxide and hydrogen ions will prove to be susceptible of reconciliation along these lines.

Deficiency of Oxygen.

Deficiency of oxygen produces hyperpnoea, even if its degree is small as compared with that caused by a rise of carbon dioxide pressure. Hyperpnoea due to deficiency of oxygen can be demonstrated without difficulty. It has been suggested that want of oxygen is associated with an increase of total acidity, chiefly in the form of lactic acid. Objections have been raised to the lactic acid hypothesis. In the first place the hyperpnoea of high altitudes could not be explained in this way. Further, it has been found that hyperpnoea occurring when the person is at rest, cannot be attributed to an increased production of lactic acid. Another hypothesis was evolved, only to be overthrown on further investigation. This was the suggestion that there was a diminished formation of ammonia in hyperpnoea. Haldane, Kellas and Kennaway postulated the following doctrine: The reduction of oxygen pressure in the alveolar air and arterial blood leads to an increase in the breathing. This is a direct result of oxygen want on the respiratory centre. The hyperpnoea is accompanied by a decrease in the alveolar carbon dioxide pressure and a reduction in the hydrogen ion concentration of the blood. In oxygen want there would be an initial alkalemia. The kidneys would react in the customary manner by excreting less acid or more alkali until the equilibrium is once again established. The compensation would not be complete. A slight alkalemia persists, which in itself would account for a reduction in the breathing. In spite of this the actual deficiency of oxygen results in an increase in the breathing. The authors of the report express the opinion that it matters little whether this effect is attributed to a stimulating property of oxygen deficiency or to an increased sensitiveness of the respiratory centre to hydrogen ions. They insist that when the respiratory centre is subjected to oxygen want, the hyperpnoea and reduction of the alveolar carbon dioxide pressure may be the accompaniments of an alkalemia and not necessarily the expression of an acidemia. In hyperpnoea caused by oxygen want there is an increase in the rate rather than in the depth of the breathing; in hyperpnoea due to increased carbon dioxide stimulation there is an increase in the depth of the breathing. In respiratory failure resulting from oxygen want there is increasing rapidity and shallowness of the breathing.

Further study has revealed the fact that increase of temperature of the blood passing through the respiratory centre renders the centre more sensitive and thus may lead to hyperpnoea.

The Effect of Exercise.

The mechanism underlying the hyperpnoea of exercise has attracted bio-physicists ever since the susceptibility of the respiratory centre to carbon dioxide has been recognized. At first it was held that the problem was purely chemical. This view was strengthened by the fact that when the carbon dioxide content of the blood was raised so that the pH of the blood increased by as little as 0.012, the total ventilation was doubled. More recently it has been discovered that the matter is far more complicated. Calculations based on the assumption of a constant value for the combined carbon dioxide of the blood, were shown to be misleading, while other sources of error upset the deductions based on other methods. Up to the present relatively little has been established concerning the production of the various forms of hyperpnoea. The authors find that proof has been provided to show that there is an actual increase in the hydrogen ion concentration of the blood during exercise and that this increase is due to alterations in the carbon dioxide, lactic and other acid contents of the blood. They are further of the opinion that, if this increase in the hydrogen ion concentration be not the cause of the increased ventilation, some other cause

consistent with a lowered carbon dioxide content of the alveolar air must be sought. It is quite essential to recognize the possibility of an important rôle being played by nervous influences. Much evidence has been collected in support of this possibility.

Dyspnoea in Certain Pathological Conditions.

The final part of the report is devoted to a consideration of the relation of breathlessness to hydrogen ion concentration of the blood in some pathological conditions. The conditions mentioned are diabetes, uræmia and renal and cardiac dyspnoea. In regard to diabetes it is shown that the earlier views are now discarded. It would seem probable that the production of ammonia in the organism in largely increased quantities in the coma of diabetes helps to neutralize the acids comprising the ketosis and to facilitate their excretion. There is obviously a slight acidemia in diabetic coma, but it is improbable that this causes the state of unconsciousness. The authors suggest that this state may be due to the poisonous character of the aceto-acetic ion. The air hunger may be due to both factors in combination.

In regard to uræmia, it would appear that there is less evidence of acid products abnormal in kind than in diabetes. An increase in the hydrogen ion concentration of the blood has been demonstrated in uræmia. By inference it would seem that in this condition there is an acidemia. In these conditions, as well as in renal dyspnoea, it is rational to assume that the breathlessness is in part at least caused by acidemia. In diabetes and in many other conditions the acidemia is insufficient to produce breathlessness or there may be no evident acidemia at all. In some of the simpler cases of cardiac dyspnoea there is a definite alkalemia. Consequently it is impossible to ascribe dyspnoea generally to one cause. The influence of nervous factors must always be remembered, even if the mechanisms by which they find their expression, may be obscure. It would seem that very little is known concerning the production of dyspnoea in pathological conditions and that the few chemico-physical facts that have been collected, need amplification before the processes can be thoroughly understood.

Medical Registration.

A CORRECTION.

OUR attention has been directed to an inaccuracy in regard to the conditions of registration under *The Medical Act of Western Australia* in *THE MEDICAL JOURNAL OF AUSTRALIA*, April 14, 1923, page 433. Clause 11 of the Act of 1894 provided that every person, male or female, shall be entitled to be registered if he or she is registered under *The Medical Ordinance of 1869*, or if he or she holds any one or more of the qualifications in the second schedule and if the diplomas, licences, certificates or other documents were obtained from some university, college or other body recognized for the purpose in the country to which such body belongs. The schedule to which reference is made, contains the following list: Fellow, member or licentiate of the Royal College of Physicians of London, of the Royal College of Physicians of Edinburgh, of the King and Queen's College of Physicians of Ireland, of the Royal College of Surgeons of England, of the Royal College of Surgeons of Edinburgh, of the Faculty of Physicians and Surgeons of Glasgow, of the Royal College of Surgeons of Ireland, licentiate of the Society of Apothecaries of London, of the Apothecaries' Hall, Dublin, doctor or bachelor of medicine or master or bachelor of surgery of some British or legally constituted and recognized Australian or New Zealand university. It further contains a clause: "Any legally qualified practitioner registered in the United Kingdom under any Act or Acts of the Parliament of Great Britain and Ireland now or hereafter in force." The last clause indicates medical officers duly appointed and confirmed of Her Majesty's sea or land services.

In brief, therefore, persons entitled to registration in virtue of British qualifications and persons actually registered by the General Medical Council in virtue of any other qualifications are eligible for registration, in addition to graduates in medicine of the Australian universities.

The fee for registration is ten guineas.

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British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held in the Lister Hall, Hindmarsh Square, Adelaide, on March 29, 1923, the President, Dr. T. G. WILSON, in the chair.

Double Nephro-Lithotomy.

DR. JOHN CORBIN showed the specimens removed from a woman, aged thirty-one years, by double nephro-lithotomy. She had been admitted to hospital on February 7, 1922, complaining of pain and swelling in the left loin. On examination a large, irregular tumour had been felt in the left loin and left side of the abdomen. A small tumour had been felt in the right kidney region. Examination by the X-rays showed a large stone in each kidney. On February 12, 1922, under ether anaesthesia, the right kidney had been exposed and a large stone was removed. There had been no pus. The left kidney had then been exposed and opened. Thick pus had been found. This had subsequently been found to be full of coliform bacilli. A large stone had been removed from the hilum. On March 4, 1922, the wounds had been healed. Dr. Corbin said that as the woman's condition had been so good after the removal of the first stone he had decided to go on and remove the stone in the other kidney. It had been his original intention only to remove the right stone at the first operation, as the kidney had not been so enlarged on this side.

Pneumoconiosis.

DR. H. CAREW NOTT exhibited a series of radiographs illustrating the various stages of pneumoconiosis from the X-ray point of view. Beginning with the earliest recognizable stage of root and peri-bronchial thickenings, the radiographs demonstrated the gradual appearance of the typical, evenly distributed mottling in the lung fields due to the small foci of lymphatic blockage. The formation of the "pleural haze" as the pleural lymphatics become blocked and congested was demonstrated. Finally the appearance of varying sized pulmonary opacities produced by fibrous masses was shown, as in the specimen exhibited by Professor Cleland.

Ossseous Abnormalities.

DR. NOTT showed another set of radiographs of a female, aged thirty-two years, who had been referred for X-ray investigation of a chronic hip joint disease. The patient had exhibited an unusual number of abnormalities. Dr. Nott demonstrated the photographs of a true cervical rib on the right side and a large seventh cervical transverse process on the opposite side; a very definite lateral spinal curvature in the mid-dorsal region, possibly congenital in origin, without any compensatory curve in the cervical or lumbar vertebrae; the twelfth rib on the left side, which was about half the size of that on the right; a mass of calcified lymphatic glands opposite the sacral promontory and, lastly, a bilateral chronic disease of the hip joints, characterized by irregular cystic bone absorption and limited by areas of bony sclerosis affecting both the femoral heads and acetabular cavities.

Silicosis of the Lung.

PROFESSOR J. BURTON CLELAND showed the lungs of a gold miner who had suffered from advanced silicosis of the lung. The patient had been a gold miner for nineteen and a half years. Acting on his doctor's advice, he had given up this occupation twelve years ago. Four years ago he had suffered from dry pleurisy, bronchitis and pneumonia. Since that time he had not been well. At the age of fifty-three years he had been admitted to the Adelaide Hospital under the care of Dr. Johnson in January, 1923. The diagnosis of silicosis suggested by the history and the clinical signs had been confirmed by skiagraphs taken by Dr. Nott. These showed extensive, densely fibrosed areas in both lungs, chiefly situated peripherally and at the left apex, increased density of the shadows at the roots of the lungs and intervening, irregu-

larly distributed streaks and small areas of fibrous tissue. No signs of cavity formation had been detected, as might have been expected had tuberculosis supervened on the silicosis. Tubercle bacilli had not been found in the sputum. At the autopsy very dense adhesions had been found in areas over both lungs. Subjacent to these there had been greyish, almost stony-hard areas of silicosis up to 7.5 centimetres in diameter. On section these areas had been as hard to cut as a dense scirrhous carcinoma of the breast. They had been situated at the apex of the left and in the middle portions of both lungs. At the root of each lung silicotic induration had also been present, though not extensive. Smaller areas of fibrosis had been scattered in other parts. The bronchial and mediastinal glands had been definitely enlarged, greyish and intensely fibrotic, a pathological condition which had extended in less degree to the glands round the celiac axis in the abdomen.

Professor Cleland said that the points of special interest were the long history of gold mining, the giving up of this occupation twelve years ago, the slow progress of the fibrosis so that definite symptoms had not manifested themselves until four years ago, the fortunate absence of complicating tuberculosis, the establishment of the diagnosis by the skiagraphs, which also suggested the absence of tuberculosis, and the location of the chief areas of silicosis at the peripheries of the lungs. Dr. Nott had suggested that this peripheral distribution of the silica particles was due to early block by silicotic reaction of the lymphatics in the roots of the lungs, with a peripheral diversion of the lymph stream. Professor Cleland thought that the adhesions over these areas suggested another possible explanation. If the adhesions had arisen during the patient's active gold-mining life, not only would they have facilitated the establishment of new lymphatic channels, but the lung, on account of being anchored by adhesions, would have been less able to clear itself of inhaled particles of silica. A greater number of these particles under these circumstances might be expected to gain entrance into the tissues of the part.

Unresolved Pneumonia, Organization and Thrombosis.

PROFESSOR CLELAND showed the lungs of a woman who had died of pneumonia at the age of fifty-five years. She had been admitted to hospital under the care of Dr. Hone, with a history of having suffered from pneumonia some months previously. On admission to hospital signs of thrombosis in the left brachial vein had been present. There had been pain on the left side of the chest, cough and signs of consolidation. Death had occurred suddenly. At the autopsy, some slight but firm pericardial adhesions had been found. The heart had been displaced to the left side by contraction of the carnified upper portion of the left lung. Only the basal portion of this lung had been crepitant, the upper part being shrunken, adherent, firm, tough and fibrous-looking, tearing into shreds, fleshy strands during removal. Microscopically the alveolar walls had appeared much thickened by increase of cellular fibrous tissue and the shrunken exudate in the alveoli had been undergoing organization. In the right lung at or near the base there had been two large, firm areas, one dark red, firm and wedge-shaped, suggesting an infarct in process of organization, the other still firmer, but pale and fibrotic, suggesting an infarct which had organized. Near the latter, on the surface, there had been an almond-sized abscess cavity, probably a localized empyema. A clot had extended from the brachial to the axillary and subclavian veins and up the external jugular vein as a softer, more recent clot. There had been no thrombosis in the internal jugular or innominate veins.

Professor Cleland said that carnification of the lung was not uncommon after lobar pneumonia. Rarely during the course of this disease thrombosis occurred in large venous trunks. In this instance thrombosis had occurred long after the acute stage, but had been associated with extensive permanent damage to the lung. It had occurred, moreover, in venous trunks in close proximity to the diseased lung tissue. Professor Cleland asked whether the site of the thrombosis was purely accidental, though due to the pulmonary disease, or had a slow inflammatory reaction spread from the adherent lung tissues to the adjacent venous walls and eventually through these to initiate the thrombosis in this manner.

Miliary Tuberculosis.

PROFESSOR CLELAND's third exhibit consisted in the lungs of a girl, aged eleven years, who had died from miliary tuberculosis of the lungs. She had been admitted to the Adelaide Hospital under Dr. de Crespigny on January 23, 1923. She had been sent to the hospital to have tonsillectomy performed, but as, on taking her temperature in routine fashion, this had been found to be 37.2° C. (99° F.), the operation had fortunately been postponed and the patient admitted for observation. The child had developed an irregular pyrexia for which no cause could be found. No abnormal signs had at first been detected in the lungs, the heart had appeared to be normal, examination of the faeces bacteriologically had failed to yield any helpful result, the leucocyte count had been nine thousand per cubic millimetre and the von Pirquet test had not yielded a reaction. On February 7, 1923, a few moist sounds had been detected in the lungs. Skiagrams taken by Dr. Nott had shown generalized miliary consolidated areas extending outwards from the roots of both lungs. These had been very suggestive of miliary tubercles. The serum had not reacted to the complement fixation test for tuberculosis. The signs in the lungs had increased and her general condition had become worse, though the patient had not complained of feeling ill. Cyanosis had finally become extreme and the respirations had reached sixty to ninety per minute. Death had occurred on February 20, 1923. The autopsy had shown both lungs to be seeded throughout with innumerable miliary tubercles. There had been small, old, caseous foci in a bronchial and in a mesenteric gland. The miliary spread had doubtless arisen from one of these.

Professor Cleland said that there were several points of interest. In the first place there was the early recognition that something was wrong with the child by the finding of a slight rise of temperature during routine examination before the performance of tonsillectomy. Had the child been operated on, the operation would unquestionably have been blamed for the lighting up of the tuberculous process. Secondly, there was the fact that the duration of the whole illness had been exactly twenty-eight days from the first recognition of a rise of temperature with absence of other signs till death. The third point was the failure of the von Pirquet and complement fixation tests to establish the diagnosis. This was probably not an uncommon feature of miliary tuberculosis. The diagnosis had been established by X-ray examination. Lastly, there was the very extensive involvement of the lungs by the miliary tubercles.

Foramen Ovale.

PROFESSOR CLELAND showed the heart of a girl, aged fifteen years, who had been admitted to the Adelaide Hospital under the care of Dr. de Crespigny. She had been delicate since birth and inclined to be languid and drowsy. Eight years ago she had had an illness during which her doctor had ordered "hot wet blankets." She had had "epileptic" fits during the last two years. She had been puffy under the eyelids in the mornings. On admission on February 15, 1923, there had been evidence of acute pericarditis, an enormously enlarged heart and physical signs suggesting the presence in the heart of a congenital defect, possibly patent *foramen ovale*. The urine had been decreased in quantity and of low specific gravity. It had contained much albumin. Maclean's test had shown a very low efficiency. The systolic blood pressure had been one hundred and ninety-five millimetres of mercury and the diastolic one hundred and forty. The radial arteries had been "leathery" to palpation. The patient in hospital had had two fits apparently of an uræmic nature. A slight improvement had taken place, but on March 2, 1923, she had developed acute pulmonary oedema and had died in a few hours.

The autopsy had shown oedematous eyelids and ankles and slight effusions into both pleural cavities. There had been a large patent *foramen ovale* and very great hypertrophy and dilatation of the left ventricle. The heart had weighed 0.46 kilograms (sixteen and a quarter ounces). This hypertrophy had seemed referable to the advanced interstitial nephritis present and its accompanying

arterio-sclerosis. The kidneys had been a little smaller than normal, had presented a somewhat coarsely granular reddish surface, had shown definite thinning of the cortex and had been in a condition of chronic parenchymatous and interstitial nephritis. The microscopical appearances had confirmed this.

Professor Cleland said that the interesting points were several. In the first place, there was the patent *foramen ovale*. In the second place there was the history of an illness, probably acute nephritis, eight years ago, as an aftermath to which had arisen the chronic parenchymatous and interstitial changes that had resulted in death. The hypertrophy of the heart was to be attributed to the renal condition, not to the cardiac defect. Consistent with the microscopic appearances of chronic parenchymatous and interstitial changes, the patient had manifested signs of both the hydræmic and the azotæmic types of renal inadequacy.

Mixed Tumour of Kidney of a Sheep (Wilson's Type).

PROFESSOR CLELAND also exhibited on behalf of Dr. L. B. BULL a tumour which had been discovered during a *post mortem* examination of a young pregnant ewe. It had not been associated with the cause of death.

The tumour was an irregular, nodular mass, situated at one pole of the kidney. It was circumscribed and showed only slight invasive tendencies at its junction with the kidney substance. There was present a large sub-capsular hæmorrhage and thrombosis of the renal vein. There were smaller diffuse hæmorrhages throughout the tumour mass, which was firm, solid, somewhat lobulated and greyish white in colour. It measured approximately ten centimetres across its large and eight centimetres across its smaller diameter.

Microscopically the tumour was seen to have an embryonal structure with cells arranged in tubular formation surrounded by masses of polyhedral and spindle cells. The differentiation between these latter cells and the more cubical or cylindrical cells arranged in tubular formation was often indistinct. There were well defined bands of fibrous tissue running throughout the section and producing a somewhat lobulated arrangement. Scattered throughout were isolated islands and columns of cartilage. The picture was typical of a Wilms's embryonal tumour of the kidney.

Psycho-Therapeutic Practice.

DR. J. M. McAREE then read a paper entitled "Some Notes on Psycho-Therapeutic Practice" (see page 543).

DR. KENNETH FRY said that he thought that Dr. McAree had really meant word association when he had described psycho-analysis. He thanked Dr. McAree for his paper and congratulated him on his results.

DR. L. L. DAVEY mentioned a patient who had suffered from epileptic fits for several years. This patient had been seen by numerous doctors and they had all agreed that he was suffering from epilepsy. He had treated the patient by suggestion and there had not been a recurrence of the fits when he had last received a report several months ago.

DR. T. G. WILSON mentioned that when he was in England last year Dr. Coué had been all the rage in London. He had been told by a leading gynaecologist that auto-suggestion was being used with great success for commencing labour. He (Dr. Wilson) asked Dr. McAree if he could explain how this phenomenon came about. He congratulated Dr. McAree on his very excellent paper and on his most interesting results.

DR. McAREE, in reply, thanked members for listening to him so patiently and expressed the hope that they might be stimulated in an endeavour to treat patients more on the lines which he had mentioned. Since his last paper a chair of psychology had been started in another State, whilst an Australian journal of psychology and philosophy was being published.

Many men had written to him about his last paper, evincing a desire to investigate for themselves. This state of affairs was very different from a few years ago, when

practical psychology and psycho-therapy was not considered ethical.

He remembered talking to a leading physician who had expressed his disbelief in Freud's sexual theory of dreams. He had admitted that he had not taken the trouble to test the matter and further added that if it were true he had no wish to know it.

If practical psychology were taught in the medical course there would not be such a bias existing in the minds of those who based their beliefs on academic systems and refused to move out of a comfortable groove.

The Medical Benevolent Association of South Australia.

SIR JOSEPH VERCO was to have addressed the meeting, but owing to his absence from the State Dr. A. A. LENDON gave a short address. (The paper prepared by SIR JOSEPH VERCO will be found on page 547).

DR. LENDON said it had not been intended that he should address the meeting that evening. Sir Joseph Verco had undertaken this duty and no one regretted Sir Joseph Verco's absence more than he (Dr. Lendon) did. Dr. Lendon's task would not be a difficult one, on account of the reasonableness of the appeal. He said that rather more than forty years ago, on August 11, 1881, the South Australian Medical Association, which had been founded in the early days of the Colony and had served its purpose, had gone out of existence in consequence of the establishment of the South Australian Branch of the British Medical Association early in the year 1880.

The old Association had made its exit quite gracefully. A small sum of money, about £150, which had belonged to the old Association had been set apart as the capital of an association with the high sounding title of the Medical Benevolent Association of South Australia. The object of this body had been primarily that of relieving distress occurring in the families of medical men and secondarily of educating their children by granting bursaries for sons who might follow their father's profession. Daughters at that time had not been contemplated as medical students. The surviving members of the demobilized association had been appointed life members. In addition to the income derived from this small amount, annual subscriptions had been invited on the understanding that all income should be used for relief, but that any balance remaining over should be added to capital. Donations other than annual subscriptions were to be regarded as capital. By thrifty management the capital had now reached over one thousand pounds sterling and an annual income of something like forty-five pounds was assured from the interest on this money. The income had been increased very little by annual subscriptions. Ten shillings was not a great amount to contribute annually and Dr. Lendon asked all present to agree to become contributors. If every member of the profession were to become a member of the fund there would be at once an income of £200 a year at the disposal of the trustees. Only about twenty members of the profession were annual subscribers. He hoped that this fact would make those present regret that the matter had not been brought properly and persistently under their notice. Small annual subscriptions were a nuisance, but the difficulty could easily be got over in two ways. Members might authorize the Secretary of the Branch to add it to their annual account or they might become life members. The sum of ten pounds not only conferred this dignity, but it added to the capital of the fund and by its interest provided the equivalent of an annual subscription.

Everyone, as Bacon had said somewhere, was a debtor to his profession. The work of this fund was truly charitable and most unostentatiously carried out. It was astonishing the number of cases of real distress that had occurred. There were instances of men who have enjoyed handsome incomes, becoming mendicants. This fate might easily be that of any member present. They did not stop to inquire whether the applicant belonged to the class of the deserving poor or the deservedly poor, for "the quality of mercy is not strained."

At the annual meeting that month one generous individual who shall be nameless, had promised £25 for ten years. Dr. Lendon asked what each member would give to back up this amount.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

- COOPER, ARTHUR GEORGE STENING, M.B., 1923 (Univ. Sydney), "Norella," Beecroft Road, Cheltenham.
CUTHBERT, NOEL MILLAR, M.B., Ch.M., 1923 (Univ. Sydney), "Elderslie," Nelson Road, Lindfield.
MARSHALL, GEOFFREY EDWIN LAMPORT, M.B., Ch.M., 1922 (Univ. Sydney), Campbell Street West, Toowoomba, Queensland (temporary address).

Proceedings of the Australian Medical Boards.

NEW SOUTH WALES.

THE undermentioned have been registered under the provisions of the *Medical Act, 1912 and 1915*, as duly qualified medical practitioners:

- FREW, CHARLES ALEXANDER, M.B., 1923 (Univ. Sydney), Eastern Hill, Albury.
GROGAN, GERTRUDE URQUHART, M.B., 1923 (Univ. Sydney), "Durham Court," Glebe Point.
GUINEY, CLARENCE MICHAEL, M.B., 1923 (Univ. Sydney), "Maisonette," Kogarah Road, Kogarah.
HALES, MARJORIE FRANCES, M.B., 1923 (Univ. Sydney), 24, Orlands Avenue, Cremorne.
HALLIDAY, JOHN HOWELL, M.B., 1923 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
HAMILTON, MARIE MONTGOMERIE, M.B., 1923 (Univ. Sydney), "Tomalib," Wentworth Road, Strathfield.
HANKINS, SYDNEY HAMILTON, M.B. (Univ. Sydney), "Alhambra," Greenwich Road, Greenwich.
HARPUR, MALCOLM DENHOLME HUNTER, M.B., 1923 (Univ. Sydney), 115, Avenue Road, Mosman.
HARRIS, RICHARD GUY SEPTIMUS, M.B., 1923 (Univ. Sydney), "Nincoola," Guyra.
HARRISON, ALFRED QUIRK OGILVIE, M.B., 1923 (Univ. Sydney), "Kelso," Martin Street, Crow's Nest.
HEWITT, GEORGE HENRY, M.B., 1923 (Univ. Sydney), "Wonga," Park Road, Burwood.
HIATT, STANLEY CHARLES MATTHEW, M.B. 1923 (Univ. Sydney), 40, Canonbury Grove, Dulwich Hill.
HOLLIDAY, ROY BOWMAN, M.B., 1923 (Univ. Sydney), "Appleby," Edward Street, Concord.
HORN, HAROLD WILLIAM, M.B., 1923 (Univ. Sydney), "Mafeking," Wellington Road, East Brisbane.
HORN, LOUIS JULIUS, M.B., 1923 (Univ. Sydney), 65, Wardell Road, Petersham.
HOTTEN, WILLIAM IVOR TOWNSEND, M.B., 1923 (Univ. Sydney), "The Hermitage," Elamang Avenue, Kirtibilli Point.
HUNT, PERCIVAL SYDNEY, M.B., 1923 (Univ. Sydney), 132, Victoria Street, Ashfield.
JEREMY, RICHMOND, M.B., 1923 (Univ. Sydney), "Elysian Flats," Milson Road, Cremorne.
KENDALL, ALBERT EDWARD HUME, M.B., 1923 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
KENNEDY, ARTHUR JAMES, M.B., 1923 (Univ. Sydney), "Camlough," Shaftesbury Road, Eastwood.
KINSELLA, VICTOR JOHN, M.B., 1923 (Univ. Sydney), "Boree," St. Mark's Road, Randwick.
KLEIN, KEITH, M.B., 1923 (Univ. Sydney), Lithgow Street, Campbelltown.
LAWES, CHARLES HENRY WICKHAM, M.B., 1923 (Univ. Sydney), 60, New Canterbury Road, Petersham.
LEVY, JACK, M.B., 1923 (Univ. Sydney), 249, King Street, Newtown.
LEWIS, BLAKE HAMMOND, M.B., 1923 (Univ. Sydney), 49, Carrington Street, Summer Hill.
LUKIN, FRANCIS WILLIAM RENNICK, M.B., 1923 (Univ. Sydney), St. Andrew's College, Newtown.
LYNCH, ALBAN JOSEPH, M.B., 1923 (Univ. Sydney), 169, Bland Street, Haberfield.
MCCREDIE, FRANK CAMPBELL, M.B., 1923 (Univ. Sydney), "Hawthorne," Angel Road, Strathfield.

MACDONALD, RODERICK HECTOR, M.B., 1923 (Univ. Sydney), Tweed Heads.

McFADDEN, AGNES SOPHIA, M.B., 1923 (Univ. Sydney), Latimer Road, Rose Bay.

MAGNUS, ALEXANDER NOEL, M.B., 1923 (Univ. Sydney), "Glenrose Flats," Elizabeth Bay.

MATHESON, WILLIAM HARLOW, M.B., 1923 (Univ. Sydney), "Woodstock," Victoria Street, Ashfield.

MILES, ERIC HILTON, M.B., 1923 (Univ. Sydney), Ware Street, Fairfield.

MONEY, REGINALD ANGEL, M.B., 1923 (Univ. Sydney), "Dalkeith," New South Head Road, Edgecliff.

MORGAN, JOHN, M.B., 1923 (Univ. Sydney), "Allerton," The Avenue, Strathfield.

MURPHY, ERIC LASCELLES, M.B., 1923 (Univ. Sydney), 44, Victoria Parade, Manly.

MURRAY-WILL, EWAN, M.B., 1923 (Univ. Sydney), Greenknowe Avenue, Potts Point.

OPIT, LEON, M.B., 1923 (Univ. Sydney), 108, Bathurst Street, Sydney.

OSTINGA, ALEX JAMES, M.B., 1923 (Univ. Sydney), "Ailsa," Creer Street, Randwick.

PARKER, DOUGLAS WILLIAM LEIGH, M.B., 1923 (Univ. Sydney), 52, Prince Albert Street, Mosman.

PONTON, RONALD GEORGE, M.B., 1923 (Univ. Sydney), "Dalkeith," Rawson Avenue, Drummoyne.

PRITCHARD, DENIS ADRIAN, M.B., 1923 (Univ. Sydney), "Harmworth," Edwin Street, Croydon.

RANKIN, ROBERT LIONEL, M.B., 1923 (Univ. Sydney), Mater Misericordiae Hospital, Lane Cove Road, North Sydney.

RAYMOND, ROLAND LIONEL, M.B., 1923 (Univ. Sydney), Kent Street, Epping.

SHEEHY, MARGARET MARY MADELEINE, M.B., 1923 (Univ. Sydney), "Bayview," Leichhardt Street, Glebe Point.

SHEPHERDSON, RUPERT FARQUHAR, M.B., 1923 (Univ. Sydney), "Kawarra," Gladstone Road, Lindfield.

SHINEBERG, SOLOMON, M.B., 1923 (Univ. Sydney), 385, Glebe Road, Glebe Point.

STEEL, ERNEST MCAUSTIN, M.B., 1923 (Univ. Sydney), "Glenstrae," Tyron Road, Lindfield.

STEEL, ROBERT STANLEY, M.B., 1923 (Univ. Sydney), "Glenstrae," Tyron Road, Lindfield.

STEPHEN, BRUCE ALEXANDER, M.B. (Univ. Sydney), "Inverkyll," Paradise Avenue, Roseville.

STORO, ALEXANDER JARVIE HOOD, M.B., 1923 (Univ. Sydney), Wesley College, Newtown.

STOBO, JOYCE SELDON, M.B., 1923 (Univ. Sydney), "Salcote," Avon Road, Pymble.

TARLETON, ARTHUR, M.B., 1923 (Univ. Sydney), 12, Wellesley Street, Summer Hill.

TAYLOR, HENRY JOHN, M.B., 1923 (Univ. Sydney), 17, Arundel Street, Forest Lodge.

THOMAS, ALFRED STRICKLAND, M.B., 1923 (Univ. Sydney), "Edinburgh," Douro Street, Mudgee.

VALLACK, RICHARD, M.B., 1923 (Univ. Sydney), 33, MacLaren Street, North Sydney.

VICKERY, COLIN EDWIN, M.B., 1923 (Univ. Sydney), Strathfield Avenue, Strathfield.

VICKERY, DONALD GEORGE ROBERTS, M.B., 1923 (Univ. Sydney), "Edina," Victoria Road, Bellevue Hill.

WALCH, JAMES HENRY BRETT, M.B., 1923 (Univ. Sydney), "Summerhome," Moonah, Tasmania.

WALTERS, CECIL JULIAN MANNING, M.B., 1923 (Univ. Sydney), Arcadia Road, Glebe Point.

WATSON, ALBERT LESLIE, M.B., 1923 (Univ. Sydney), "Magnolia," Lansdowne Street, Eastwood.

WISE, ALLEN GLENDENNING, M.B., 1923 (Univ. Sydney), Florence Street, Strathfield.

WITTS, FANNIE EVA, M.B., 1923 (Univ. Sydney), 20, Musgrave Street, Mosman.

WOODS, JACK MCKENZIE, M.B., 1923 (Univ. Sydney), 37, Roslyn Gardens, Darlinghurst.

Medical Appointments.

DR. W. P. GALLAGHER (B.M.A.) has been appointed District Medical Officer and Public Vaccinator at Quairading, Western Australia.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30-34, Elizabeth Street, Sydney	Australian Natives' Association Ashfield and District Friendly Societies' Dispensary Balmmain United Friendly Societies' Dispensary Friendly Society Lodges at Casino Leichhardt and Petersham Dispensary Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney Marrickville United Friendly Societies' Dispensary North Sydney United Friendly Societies People's Prudential Benefit Society Phoenix Mutual Provident Society
VICTORIA: Honorary Secretary, Medical Society Hall, East Melbourne	All Institutes or Medical Dispensaries Australian Prudential Association Proprietary, Limited Manchester Unity Independent Order of Oddfellows Mutual National Provident Club National Provident Association
QUEENSLAND: Honorary Secretary, B. M. A. Building, Adelaide Street, Brisbane	Brisbane United Friendly Society Institute Stannary Hills Hospital
SOUTH AUSTRALIA: Honorary Secretary, 12, North Terrace, Adelaide	Contract Practice Appointments at Renmark Contract Practice Appointments in South Australia
WESTERN AUSTRALIA: Honorary Secretary, Saint George's Terrace, Perth	All Contract Practice Appointments in Western Australia
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington	Friendly Society Lodges, Wellington. New Zealand

Diary for the Month.

MAY 22.—New South Wales Branch, B.M.A.: Medical Politics Committee; Organization and Science Committee.

MAY 24.—Brisbane Hospital for Sick Children: Clinical Meeting.

MAY 25.—New South Wales Branch, B.M.A.: Branch.

MAY 25.—Queensland Branch, B.M.A.: Council.

MAY 30.—Victorian Branch, B.M.A.: Council.

MAY 31.—South Australian Branch, B.M.A.: Branch.

JUNE 1.—Queensland Branch, B.M.A.: Branch.

JUNE 6.—Victorian Branch, B.M.A.: Branch.

JUNE 8.—New South Wales Branch, B.M.A.: Clinical Meeting.

JUNE 8.—Queensland Branch, B.M.A.: Council.

JUNE 8.—South Australian Branch, B.M.A.: Council.

JUNE 12.—New South Wales Branch, B.M.A.: Ethics Committee.

JUNE 13.—Western Australian Branch, B.M.A.: Council.

JUNE 13.—Melbourne Pediatric Society.

JUNE 14.—Victorian Branch, B.M.A.: Council.

JUNE 15.—Eastern District Medical Association, New South Wales.

JUNE 19.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

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